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Archives of Neurology and Psychiatry

Vol. 4 SEPTEMBER, 1920 No. 3

ELEMENTS OF PREDISPOSITION AND DETERMINING CAUSES OF SECONDARY DISORDERS IN PSYCHO-NEUROTIC ACROPARALYSIS RESULTING FROM THE WAR: "THE DYSKINETIC SYNDROME"

GUSTAVE ROUSSY, M.D.

PARIS

Among the numerous nervous disorders brought to light by the war, there is a syndrome with peculiar clinical manifestations which has impressed the French neurologists (Meige, Pierre Marie, Foix, Babinski, Roussy, Boisseau and d'Oelsnitz). This syndrome is characterized by contractures resulting in fixation of the larger articulations (hips, shoulders, elbows and knees) in defective postures, and by motor difficulties of a special type—contraction, paralysis or an association of both—localized in the distal ends of the limbs and producing a condition described by the French as "main figée" (palsied stiff hand) and "pied bot réflexe" (reflex club foot).

Following a nervous shock caused by the bursting of a shell in close proximity, or traumatism, often slight, or certain other causes, a state of impotency, dependent on a more or less well developed pithiatic character, becomes manifest. At the end of a period of time, which varies in the different cases, the influence of immobility or defective function, somewhat conditional on certain predispositions (circulatory, etc.) leads to secondary organic accidents, lasting as long as the immobility itself; these, however, gradually decrease and eventually disappear after an indefinite length of time, if the normal movements are restored.

The question, therefore, is no longer that of mere pithiatic accidents, but really of hystero-organic associations. Consequently two elements of the problem to be considered are: (1) the chief motor disorder, (2) the secondary disorders (vasomotor, trophic, etc.), which develop later.

I shall attempt at this time, without reference to the extended series of reports that have been published in France on this subject, to recapitulate the different signs and symptoms which constitute this syndrome.

MOTOR DISORDER

Motor disorder (contraction or paralysis) has long been considered obstinate when treated by psychotherapy. I have demonstrated, however, with Boisseau and d'Oelsnitz that it usually assumes the characteristics of a pithiatic accident, namely, it can be made to recur or disappear suddenly by persuasion. I also stated in previous publications that the posture, dependent on paralysis or contracture, does not seem at first sight to be capable of reproduction on the normal side by the patient. However, it is possible to demonstrate that a state of individual articular or musculotendinous laxity may be present, enabling the patient to assume attitudes not normally possible.

SECONDARY DISORDERS

Secondary disorders are the clinical characteristics of the organic accidents which Babinski and Froment first observed and minutely described. We shall choose, as a standard, those accidents that are described as "stiffened hands," in which the complete array of signs and symptoms constituting this syndrome may be observed.

Vasomotor and Thermic Disorders.—The skin of the hand and sometimes of the forearm as well, is cyanotic, the color varying from bluish or purple to salmon red. The surface temperature may be from 2 to 6 degrees (centigrade) less than that of the normal hand. By means of a Pachon sphygmomanometer it is easy to discern that the arterial oscillations, especially during cold weather, are less in amplitude on the affected side (Babinski and Heitz). These authors have shown, moreover, that after a bath of five minutes' duration at 40 C., the affected hand is warmer, and the arterial oscillations of greater amplitude than on the healthy side. Therefore arterial lesions may be excluded as a cause of the cyanosis. Moreover, I have observed, with Boisseau and d'Oelsnitz, that sudden restoration of active movements after treatment produced the same result (disconnecting test). Furthermore, I have pointed out that on the healthy side of such patients, there were more or less obvious vasomotor disturbances, showing that they, possibly on account of a congenital predisposition, have vasomotor disturbance. This fact proves that the patients, possibly on account of a congenital predisposition, do not possess a vasomotor system which functions normally, and that, therefore, the marked changes on the affected side are more easily produced by factors dependent on immobility than in normal persons.

Mechanical Overexcitability of the Muscles.—By tapping the muscles of the hand or arm of the affected side with the finger, the muscular contraction produced is stronger and of longer duration than on the

healthy side. The electrical excitability of the muscles and nerves may be modified likewise, although a reaction of degeneration is never present.

Disorders of Sweat-Secretion.—Hyperhidrosis (over-sudation) is often present on the affected side, sometimes so marked as to produce sudamina. I have pointed out that this disorder existed in patients in whom a tendency to excessive sweating was also present on the normal side.

Trophic Disturbances.—These are often well developed. The skin becomes thin, smooth and glossy. The wrinkles are obliterated. When the vicious attitudes are of long duration, genuine cutaneous retractions may occur. In not a few cases the skin is infiltrated and appears "succulent." The nails become hard, brittle and show transverse or longitudinal striations. Occasionally they are convex, hippocratic and deformed. They often grow faster than on the normal side. The hair is longer and thicker than on normal side, or on the contrary, broken, smaller and less abundant.

Some muscular atrophy, generally slight, may occur. Occasionally, however, the circumference of the arm may be from 4 to 6 cm. less than that of the normal side. Tendinous contractures are often present in long standing cases. The phalangeal and metacarpophalangeal articulations are frequently stiff and painful and at times even fibroankylosis is present. Nodular deformities, such as are found in arthritis, may occur. Even the bones may be so altered that decalcification and osteoporosis is revealed by roentgen-ray examination.

Modification of the Tendinous and Cutaneous Reflexes.—Babinski and Froment have shown that the tendon reflexes may be exaggerated on the affected side and that this increased excitability becomes even more pronounced when the patient is anesthetized by chloroform. With Boisseau and d'Oelsnitz, I have demonstrated for the first time the relation between this hyperreflectivity and muscular atrophy. The ankle reflexes are diminished (Vincent) and plantar response by plantar stimulation may be abolished (Babinski and Froment). These modifications occur in instances in which cyanosis and hypothermia are prominent features. They disappear as soon as the limb becomes warm.

Muscular Hypotonicity.—This may be so marked in certain instances that the palmar surfaces of the fingers may touch the anterior surface of the forearm.

The association of the motor disorders with the secondary disorders just described forms a peculiar clinical syndrome, whose localization in divers segments of the limbs gives rise to a series of psycho-

neurotic accidents which cannot be completely detailed within the limits of this article. I shall, however, mention a few of the more frequent ones.

I have chosen as the typical case the "stiffened hand" because in such instances secondary accidents are not only common and as a rule well developed, but also because this condition has been studied with special care. In foot palsies and acrocontractures (psychoneurotic club-foot, clutched toes) and in psychoneurotic contractures of the elbows, knees, hips and shoulders, these secondary phenomena are

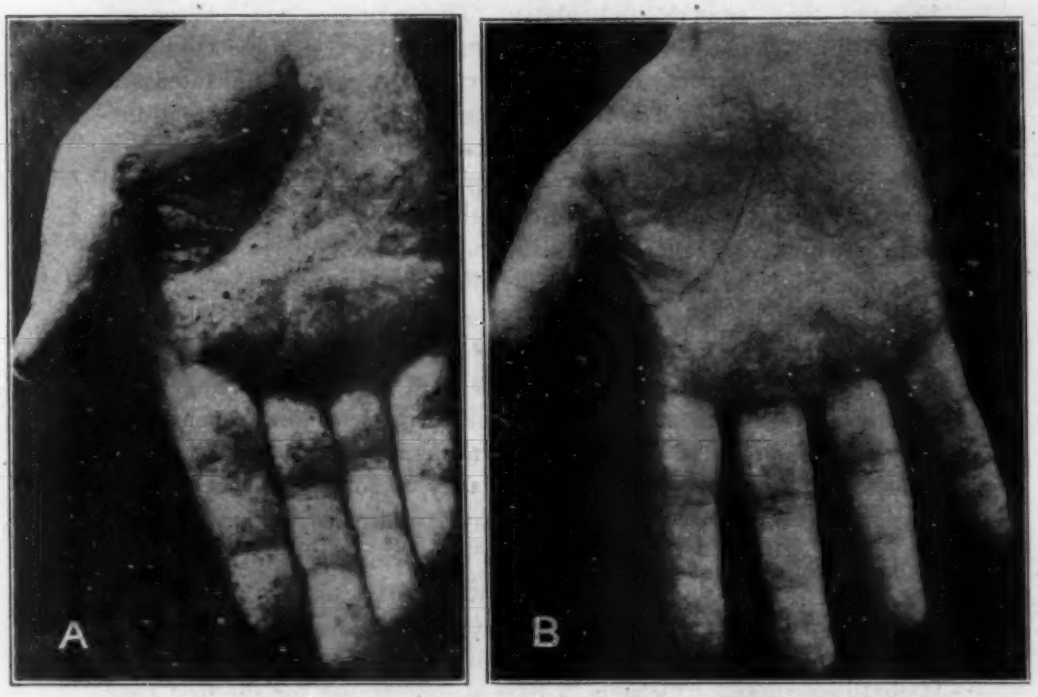

Fig. 1.—Hand from a case of contracture of the elbow and difficulties of flexion of the first phalanx of the third and fourth fingers, with fibrous formations and limited function, eleven months after the injury in the collapse of a dugout; *A*, showing numerous sudamina caused by hyperhidrosis; *B*, disappearance of cutaneous disturbances, including hyperhidrosis, after the recovery of motor function.

often present. They may occur not only in palsies and contractures of segments of the limbs, but also in some instances of monoplegia or pithiatic paralysis.

Nature of Vasomotor and Trophic Disturbances.—In what group do the disorders just described belong? Are they hysterical or organic? These questions, often discussed during the war, have not been unanimously and finally solved. Without entering into details,

I shall mention briefly the principal theories that have been advanced in France as an explanation of their pathogenesis.

THE REFLEX THEORY: According to Babinski and Froment, these accidents fall into a special group "intermediate between the strictly organic and the hysterical disorders." They attribute the origin of these disorders to reflex causes and insist, in their later reports, on the frequency of the association and the progressively predominant influence of the hysterical element, sometimes amounting to the complete replacement of the pithiatic element.

THE DYNAMOGENIC THEORY: Claude and Lhermitte admit that in the beginning of the functional motor disturbances, an organic lesion (slight wound) may, in certain instances, operate by a reflex mechanism. But—and here they differ from the former theory—this reflex mechanism (originally causal?) plays only a short and transitory rôle. If treated properly, the disorders will disappear without leaving any residuum (circulatory, articular, trophic), but if not so treated, the conditions become fixed by a psychic mechanism, and their constant presence produces dynamic modifications of the nervous centers, as shown by the study of motor, sensory and reflex phenomena during the narcotic state.

THE DYSKINETIC THEORY: (*The Immobility or Defective Action Syndrome.*—Under this title, in cooperation with Boisseau and d'Oelsnitz, I have grouped the ideas that we have expounded before. The basis of our conception, supported by and founded on the careful clinical observations of more than 2,000 cases of war psychoneuroses, may be stated thus:

1. The motor disorder, contraction or paralysis, is the principal and primary element of the syndrome; it is also pithiatic in character and consequently capable of reproduction at the will of the patient. It is liable to disappear suddenly under the influence of persuasion if this method is employed under favorable conditions.

2. The associated disorders, which are commonly but not constantly present, are variable in character and very persistent. They are, according to Babinski's classic conceptions, comparable in every way to disorders admittedly hysterical in character. They are, furthermore, functional accidents that have suddenly appeared in patients predisposed to pithiatic motor disturbances.

Can our conceptions of their pathogenesis be made to agree with the facts observed? We believe they can and I shall therefore recapitulate certain arguments in support of our opinion.

It is well known that the secondary disturbances do not appear indifferently in every instance of pithiatic motor disorder. What, therefore, are the causes and laws on which this selective association

depends? As the result of our investigations we are inclined to believe that the origin of these secondary disorders is doubly conditional and dependent: (1) on elements of constitutional or acquired predisposition antedating the pithiatic motor disturbances, and, (2) on determining causes operating for a longer or shorter time on an adequate predisposition and producing completely the array of secondary disorders.

PREDISPOSITION

The systematic observation of the healthy half of the body of patients affected by neurotic disorders has led to certain interesting conclusions. The most typical and complete example is that of the "main figée." Its clinical characteristics are: hypothermia and cyanosis are present; oscillometry demonstrates more or less marked diminution of the arterial oscillatory amplitude; sudoral disorders are present; the reflexes are modified; and there are deformities, grotesque in appearance and apparently not possibly produced by the will or by an hysterical condition. We have constantly attempted to discover whether these various alterations were not also present in a larval or latent state on the unaffected side, and our efforts in this direction have not been unrewarded.

Repeated examinations of the minor alterations of function on the healthy side have demonstrated that the same tendencies are present but of lesser degree. Therefore, the same predisposition to pithiatic disturbances has been identified in both limbs but at different functional levels. I shall now describe these phenomena and later discuss their significance.

The Circulatory Phenomena, Microsphygmia.—Oscillometric investigation of the numerous cases of acromotor disorder has disclosed that the healthy side of the patient is in a condition of vasoconstriction not so marked as on the affected side, but definite as compared with the results obtained on normal subjects. This condition is sometimes appreciable by sight due to the acrocyanosis. We have verified this observation not only by repeated readings with the Pachon oscillometer, but also by graphic records or "oscillograms." It is apparent, therefore, that particular stress should be placed on the existence of bilateral microsphygmia in patients with "main figée" and other pithiatic disturbances.

The Sudoral Phenomena.—The systematic studies of the healthy side under different conditions have demonstrated certain other tendencies. If a hand is perspiring so abundantly that it seems glossy and beads of sweat appear on the palmar surface, the same tendency to hyperhidrosis, less marked but still abnormal, will be observed on the other hand.

Articulo-Musculo-Tendinous Laxity.—If extreme hyperextension of the fingers is present, apparently not produced by the will or even by an hysterical disorder of the affected side, decidedly abnormal musculo-tendinous laxity will be found on the healthy side. It is under these conditions that antalgic attitudes, often voluntarily fixed, are apt to occur. The deformities so produced at first sight seem to exceed the limits of purely hysterical disturbances.

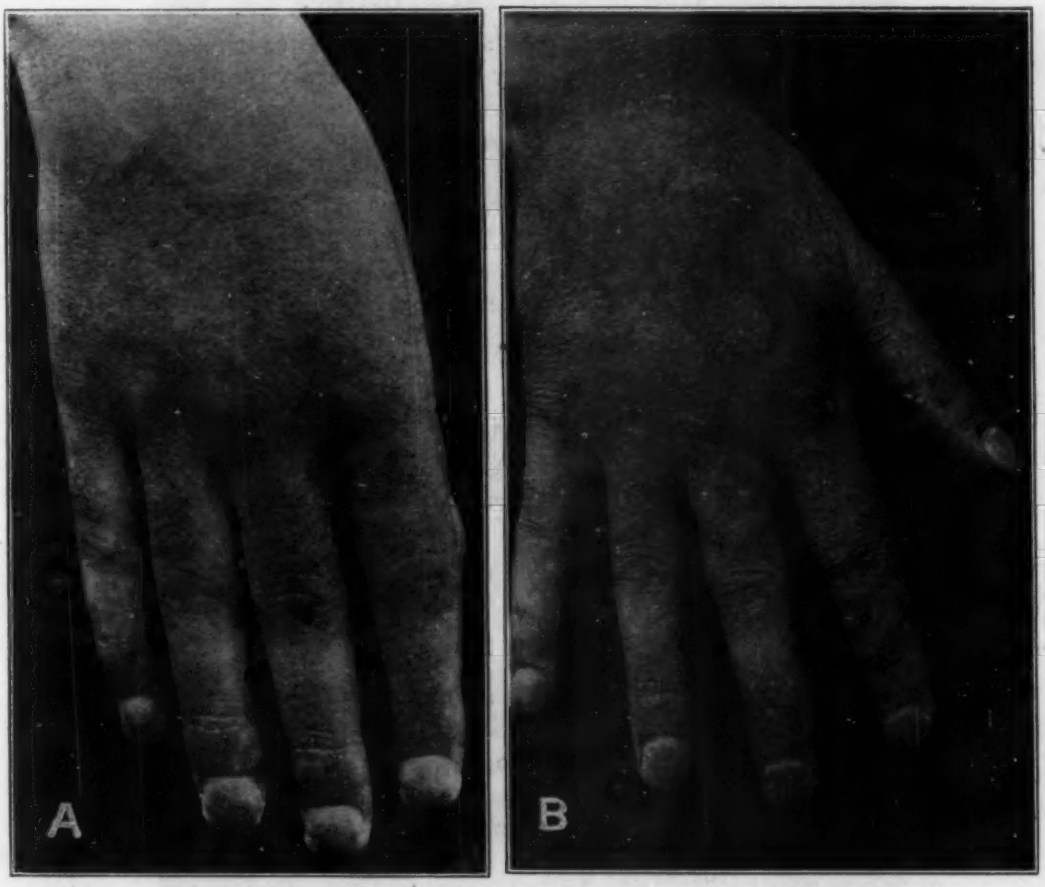

Fig. 2.—Right hand paralyzed (main figée), thirty-five months after penetrating gunshot wound of the forearm; *A*, showing trophic disorder of the nails (longitudinal incurvation) and of the skin (glossy appearance) with disappearance of the wrinkles around the knuckles; *B*, reappearance of the wrinkles after several weeks, during which the patient has regained the use of his hand.

ARTICULAR DISORDERS

Why are certain persons subject to early articular stiffness while others show no signs of the disturbance even after a long period of immobility? Although immobility is necessary for the production of

articular disorders, alone it is not a sufficient cause. A variable association of predisposing causes is required. In certain instances long suppuration appears to promote articular reactions, in others former articulatory disturbances bring up the question of a diathetic predisposition. Immobility and predisposition therefore appear to be necessary and sufficient for the origination and development of the articular symptoms.

TENDINOUS HYPERREFLECTIVITY

Should clinical observation detect a state of excessive tendinous reflectivity, the healthy side will usually reveal a tendency toward strong reflex reactions. This has been proved daily during clinical investigations. How may we explain this amplification of the tendinous reflexes? It is noteworthy, as stated above, that the coexistence of hyperreflectivity and muscular atrophy has frequently been observed. Moreover, in a number of patients a slow but progressive diminution of tendinous hyperreflectivity has been observed coincident with the amelioration of the motor disorder and paralleling the resumption of normal function, and consequent subsidence of muscular atrophy in the affected limb.

I believe, therefore (taking into account the results obtained by Babinski and Froment from the study of the reflexes in a prolonged narcotic state, and without rejecting Claude and Lhermitte's ingenious hypothesis of the dynamogenic action operating through the long inactivity of the limb on the medullary centers), that another hypothesis can be advanced. Cannot the reflex modifications be caused (in certain cases, and to a greater or less degree) not by an anatomic or dynamic alteration of the center (spinal cord) of the reflex arc (conductors, nerves) but by an affection localized at the starting point of the arc —the muscles? In this instance the tendinous hyperreflectivity might be connected with the anatomic (histochemical) modifications and thus with the physiologic modifications of the muscle fiber itself (myofibrilla or sarcoplasm).

This consideration of the abnormalities observed on the healthy side in hysterical patients has made it possible to perceive dimly, in their larval state, most of the disorders of the affected side. But are alterations found that antedate the morbid disturbances (genuine elements of predisposition) rather than the direct influence of the affected side on the normal side? I believe so. The disorders of the healthy side cannot, when subjected to early and repeated observation, be explained by the laws of symmetry; and moreover we usually notice that, according to their presence or absence, the same neuropathic motor troubles will or will not bring about morbid aggravations on the side affected.

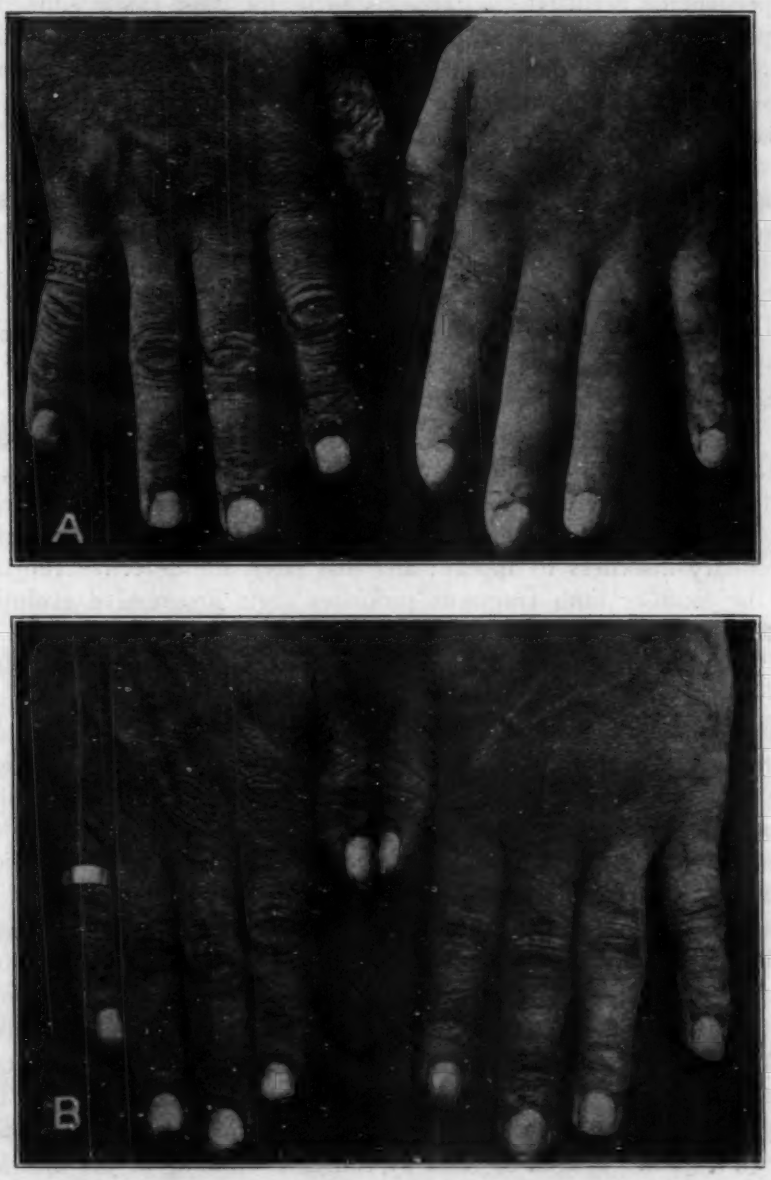

Fig. 3.—Paralyzed left hand (in extension), twenty-seven months after a fistulous bullet wound of the forearm, with reflex disorders; *A*, showing the trophic involvement of the skin and nails of the left hand, as is clearly discernible by comparison with the right hand; *B*, reappearance of the wrinkles around the knuckles following marked improvement several weeks after the restoration of function.

The preexistence, therefore, of bilateral and symmetrical disorders dependent on predisposition appears to be the most easily admissible hypothesis to explain our observations. Hence, the most plausible hypothesis is that the full development of the secondary syndrome observed on the affected side is only an abnormal amplification of preexisting tendencies observed on the healthy side.

But what are the circumstances that have brought about such an amplification or accentuation and have thereby converted a manifestation that is physiologic into a disorder obviously pathologic?

DETERMINING CAUSES

We may now observe the intervention of two essential factors: the influence of immobility or defective function and the influence of the pyschic state of the patient.

The Influence of Immobility or Defective Function.—I believe that antalgic immobility through psychic inertia, following traumatism, as well as immobility obtained by compression in any fracture—reducing apparatus—is enough to aggravate the preexisting tendencies and cause secondary disorders to appear; and that later, the defective function of the limb or limb fragment promotes their progressive evolution and development.

Thus the different disorders will occur in this chronological order: the caloric and vasomotor conditions with their sequelae (mechanical hyperexcitability, modifications of the electric reactions); the trophic disturbances, amyotrophia (with consequent hyperreflectivity), articular fibro-ankylosis and musculotendinous retraction. If immobility alone does not generally suffice to give rise to circulatory disorders which preexist, it may, when associated with previous vasoconstriction, occasion them. Thus immobility and vasoconstriction, though of independent origin, are factors which undergo a parallel evolution and add their effects to produce the syndrome in its symptomatic whole.

My conception of the mechanism of its production is, therefore, that the bilateral acrovasomotor disorders are themselves dependent, even before any traumatic or dyskinetic element appears, on a certain degree of irregularity in the vascular sympathetic system. They betray a deficiency in the thermic and vasomotor functions against which the patients are obliged to react by constant straining of the muscles. The intensity and frequency of the movements make up for the retardation of the circulation by exciting the inadequate functions of the sympathetic system. Under these circumstances, when a limb, or rather a limb end, in which such troubles are found, lies motionless under direct (antalgic) or indirect (pithiatic) influence of some local traumatism, what will occur? "The organs"—to quote Morat and

Doyon—"produce, through the medium of their sensory nerves, the vasomotor phenomena destined to equalize the circulation to the need occasioned by the effort."

The muscular inactivity will, therefore, increase the inhibition of an already inefficient sympathetic system. The longer this inactivity lasts, the more accustomed the sympathetic system becomes to the new state of things and it acquires a decided tendency to lose completely its normal regulatory functions. What happens if the motor functions of the limb are made to reappear?

Passive mobility will only modify for a time, and in a very limited manner, the vasomotor conditions; this is accounted for by the fact that passive motion is not a revival of the muscular functions but only a false appearance of the physiologic action of the muscles. Consequently there is no reason to expect it to influence the sympathetic centers in any important or lasting manner. On the contrary, the recurrence of active movements causes, as has been shown, an intense modification with remarkable improvement of the vascular disorders. If the result does not prove complete and final, if it shows a tendency to stabilize gradually, it is, in my opinion, because the sympathetic system of the limb has grown accustomed to muscular inactivity and because the adaptation to renewed activity requires regular and continual exercise of the muscles in their normal physiologic function.

It is therefore my opinion that immobility leads to the appearance of secondary disorders in predisposed persons. It is the suppression or the defectiveness of their functions that allows the disorders to persist. Restoration of activity leads to improvement and physiologically normal exercise is likely to cause the disorder to disappear.

Necessity of a Special Mental State.—A persistent, systematic and even unnatural cessation of function is necessary to produce such disorders. The patient apparently does all in his power to lose the use of his limb or limb fragment. The patients are persons with a special turn of mind, whose will is deficient or more or less perverted, reminding us in many particulars of that of patients injured in industrial accidents. This state of mind is an essential etiologic factor necessary for the growth and development of the disorders considered. It enables them to appear, explains their characteristics and furnishes rules to be followed in their treatment. The mental state must be always taken into serious consideration in deciding the prognosis and treatment of a psychoneuropathic accident.

SUMMARY

After an investigation of more than 2,000 psychoneurotic disorders, these conclusions appear to be justifiable:

1. To enable the appearance of vasomotor and trophic disorders, during the course of acrocontractural and acroparalytic manifestations, three conditions are necessary: (*a*) Elements of predisposition and among them circulatory troubles, (*b*) immobility or defective function, and, (*c*) a special mental state.

2. The circulatory caloric and trophic troubles are only manifestations of a secondary class arising from a primary condition of neuropathic nature, paralysis or contracture.

I have therefore, proposed to designate the whole series of disorders constituting this syndrome "the dyskinetic syndrome, or syndrome of defective function."

PERIPHERAL NERVE TOPOGRAPHY

SEVENTY-SEVEN OBSERVATIONS OF ELECTRICAL STIMULATION OF NORMAL AND DISEASED PERIPHERAL NERVES

WALTER M. KRAUS, A.M., M.D.

NEW YORK

AND

SAMUEL D. INGHAM, M.D.

PHILADELPHIA

1. Introduction
2. Technic
3. Material
4. Levels of Stimulation
5. Electric Phenomena
6. Plexus Formations in Nerves, Fasciculi and Fibers
7. Preponderance of Reaction in Proximal Muscles
8. Straight Course of Motor Fasciculi
9. Findings in Individual Nerves and Their Branches
10. Case Reports and Charts

INTRODUCTION

The large number of injuries to peripheral nerves which occurred during the world war centered the attention of many on the details of their anatomy. It became important in considering partial lesions to know the functions of the various fasciculi that made up a nerve. Clinical examination often indicated an incomplete lesion, and a study of the course of the bullet or piece of shrapnel showed that one or another aspect of the nerve trunk had been the main site of the injury. In other cases compression by scar tissue affected the nerve function. When the case reached the stage at which operation was performed, the question sometimes arose—What relation is borne by the clinical to the operative findings? Does an external lateral neuroma mean that certain muscles must be paralyzed, and does this coincide with the clinical findings? Figure 1 from Dustin's [1] paper gives a graphic idea of this problem. In addition to these questions, others of more theoretical value arose. The whole problem of the anatomic basis of physiologic facts or theories relating to sensation and the conduction of stimuli to muscles in the peripheral nerve made its appearance.

1. Dustin: La fasciculation des nerfs, in Ambulance de "L'Ocean," **2**:pt. 1, 135 (July) 1918.

All this resolves itself into the question—What physiologic significance has each intraneural bundle of fibers or fasciculus? Is it motor, and if so, what muscle does it innervate? Are sensory fibers included in motor fasciculi and how do they reach them? Which fasciculi are purely sensory? When segments contribute to a nerve, where do their various fibers rearrange themselves? The questions multiplied. It became clear that we had little knowledge of the physiologic aspect of the numerous fiber tracts (fasciculi) contained in the various nerves. Compared to our knowledge of spinal cord tracts, that of peripheral nerve tracts was extremely meager.

Stoffel,[2] in 1911, did some thorough work on this subject. By gross dissection, he succeeded in separating a large number of fasciculi, in determining the gross appearance on cross section of many of the larger nerve trunks and in correlating these pictures with the functions of the various fasciculi appearing in them. He correlated the

Fig. 1.—Involvement of fasciculi from a lateral neuroma (from Dustin).

transverse as well as the longitudinal pictures in terms of the location and function of the fasciculi.

The next work was done in France and concerned war injuries. Marie [3] and his co-workers, by means of electrical stimulation of nerves exposed at operation, confirmed some of the conclusions of Stoffel and added new facts. They correlated the cross section work of Stoffel with the findings they obtained by stimulation of the circumference of the nerves and found that they agreed in many instances.

In England, Langley and Hashimoto,[4] using a third method—dissection of macerated nerve trunks—made contributions to the subject, mainly in regard to the interlacing of the fibers of various fasciculi, i. e., *internal nerve plexuses.*

Dustin [1] of Brussels, in July, 1918, did a large piece of work by gross dissection and by correlating the appearance of successive cross-

2. Stoffel and Vulpius: Orthopëdische Operationslehre, 1911.

3. Marie, Meige, Gosset and Pascals: Les localizations Motrices dans les nerfs peripheriques, Bull. de l' Acad. de méd., Par. **74**:798, (Dec. 28) 1915.

4. Langley and Hashimoto: J. Physiol. **51**:318, 1917.

sections of the nerves. His article contains illustrations showing the cross sectional appearance of the various parts of the brachial plexus and the median nerve; but the work is purely anatomical and, though of importance, led to no correlation with the functions of the fasciculi he describes.

Therefore, by the end of 1918, considerable work of various kinds had been done. The present investigation is a continuation of that of Marie, and has the same aim in view, namely, to determine, by application of a faradic current to the circumference of the nerve, the location and function of the fasciculi that lie beneath.

TECHNIC

This article is based on work done on nerves, normal and abnormal, exposed at operation in the U. S. army hospitals at Cape May, N. J. and Fox Hill, N. Y., in 1919. The difficulties of investigation are not many if care is taken to follow a few simple procedures.

Orientation of the Nerve.—The various anatomic aspects of the nerves must be properly labeled to permit ready recognition at any time. The details of this and of the technic at operation are minutely described in a previous article.[5]

MATERIAL

Of the cases in which an attempt was made to localize bundles, only those that gave a positive result are included. Approximately 25 per cent. of the cases tried gave a negative result, due to complete functional interruption of the entire nerve. The positive cases, fifty-eight in number, on which seventy-seven observations were made, are charted in Table 1.

The positive cases reported by Marie, Meige and Gosset numbered forty-three. Déjerine and Mouzon [6] did not cite the number of cases observed, but merely stated their findings in various nerves. Neither recorded the individual cases.

The patients in this series had been injured at least six months prior to examination so that degeneration was completed long before they presented themselves. Most of the cases had been at a standstill for a considerable period, and were therefore deemed ready for operative interference. One case (Case 49) was in a stage of partial regeneration after a previous suture.

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5. Kraus, W. M., and Ingham, S. D.: Electrical Stimulation of Peripheral Nerves Exposed at Operation: "Surgical Value," J. A. M. A. **74**:586 (Feb. 28) 1920.

6. Déjerine, M. and Mme., and Mouzon: Les lesions des gros troncs nerveux des membres par projectiles de guerre, Presse Méd., May 10, July 8 and August 30, 1915.

TABLE 1.—Number of Reactions in Nerves and Muscles in Fifty-Eight Operative Cases

	Case Numbers	Nerve	Muscles and Sensations	Total
I. Brachial plexus	5, 41	5		
1. Pectoralis major		..	1	
			—	1
II. Median nerve:				
(a) Outer head	31	1		
(b) Inner head	11, 31	2		
(c) Trunk	2, 3, 9, 13, 16, 17, 19, 26, 27, 30, 36, 43, 44, 45, 50, 53, 67, 69	18		
1. Pronator radii teres		..	14	
2. Flexor carpi radialis		..	12	
3. Palmaris longus		..	3	
4. Flexors of the fingers		..	11	
5. Flexor longus pollicis		..	7	
6. Pronator quadratus		..	2	
7. Abductor pollicis		..	2	
8. Opponens pollicis		..	1	
9. Lumbricales		..	1	
10. Sensory		..	3	
			—	56
III. Ulnar nerve	3, 8, 11, 13, 16, 18, 27, 28, 31, 34, 35, 42, 43, 45, 47, 49, 50, 60, 66	19		
1. Flexor carpi ulnaris		..	15	
2. Flex. profundus digitorum		..	13	
3. Adductor pollicis		..	6	
4. Interossei		..	5	
5. Hypothenars		..	4	
6. Sensory		..	1	
			—	44
IV. Circumflex nerve	5, 31, 39	3		
1. Deltoid		..	3	
			—	3
V. Musculospiral nerve	15, 22, 31, 33	4		
1. Triceps		..	4	
2. Supinator longus		..	2	
3. Extensor carpi radialis		..	2	
4. Extensors of the fingers		..	1	
			—	9
VI. Sciatic nerve:				
(a) Internal popliteal divis.	12, 20, 23, 24, 64	5		
1. Hamstrings		..	3	
2. Gastrocnemius		..	4	
3. Tibialis posticus		..	3	
4. Flexor longus digit.		..	2	
5. Flex. longus hallucis		..	2	
6. Intrinsics		..	0	
			—	14
(b) Ext. popliteal division.	14, 21, 59	3		
1. Peronei		..	3	
2. Tibialis anticus		..	2	
3. Extensor digitorum.		..	1	
4. Extensor hallucis		..	1	
			—	7
VII. Internal popliteal nerve	1, 6, 7, 10, 40, 51, 52, 54, 56, 58, 63, 64, 68	12		
1. Hamstrings		..	1	
2. Gastrocnemius and soleus		..	9	
3. Tibialis posticus		..	10	
4. Flexor longus digitorus		..	5	
5. Flexor longus hallucis		..	4	
6. Intrinsics		..	2	
			—	31
VIII. External popliteal nerve	10, 14, 38, 46, 59	5		
1. Peronei		..	5	
2. Tibialis anticus		..	2	
3. Extensor digitorum		..	1	
4. Extensor hallucis		..	1	
			—	9
Total		77	..	174

LEVELS OF STIMULATION

The key to the level of stimulation of the different nerves may be found in the diagrams (Figs. 2 and 3). It will be seen that the various nerves have been stimulated at practically *all levels*. The case reports are arranged in order of the level of stimulation beginning at the proximal part of the nerve. The stimulation was applied to the nerve trunks a little below and a little above the lesions, as well as on the injured portions of the nerves.

ELECTRIC PHENOMENA

The electric phenomena which were brought to light by this work have been briefly commented on in our previous paper.[5] These phenomena were observed:

1. A phenomenon of difference between direct and percutaneous stimulation, fourteen cases (2, 3, 5, 8, 9, 15, 21, 24, 26, 31, 36, 39, 43, 49). The importance of this phenomenon when operating on nerves is clear. When a fasciculus, which is inactive on stimulation through the skin, is found to be active on stimulating the nerve directly, its conductivity is proven and should not be cut. The converse, that absence of any reaction indicates no live fibers, is not necessarily true for obvious reasons. This phenomenon must also be interpreted in each case in terms of the following one.

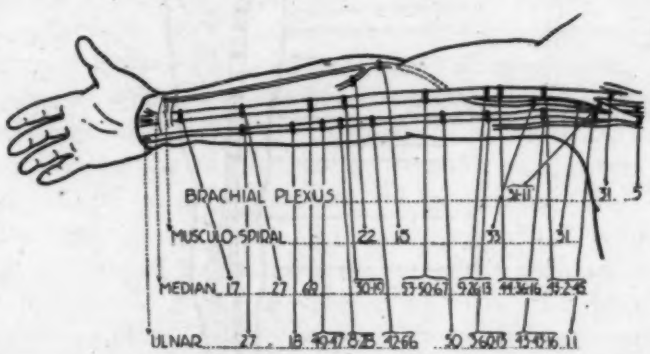

Fig. 2.—Level of stimulation of the principle nerves of the arm and their branches.

2. A direct Erb's paradoxical phenomenon. In this condition some muscles reacted when the nerve was directly stimulated *below* the lesion, which did not react when the nerve was directly stimulated *above* the lesion. Thus the lesion acted as a delaying agent for the current (Cases 13 and 28).

A discussion of the significance of these two phenomena for nerve physiology would consume too much space. Their practical importance requires no discussion.

PLEXUS FORMATIONS IN NERVES, FASCICULI AND FIBERS

The question of internal nerve plexuses is a crucial one in the problem of fascicular anatomy and physiology.

In monosegmental mixed nerves, the deep branches (to muscles) contain, besides motor fibers, those carrying proprioceptive sensations and pain. Since these sensory fibers are of posterior root origin while the motor fibers are of anterior root origin, it is obvious that there must be an *internal nerve plexus* due to the change from the condition of root fasciculi at that of nerve fasciculi. Sensory fibers must pass to motor fasciculi. This demands one type of internal nerve

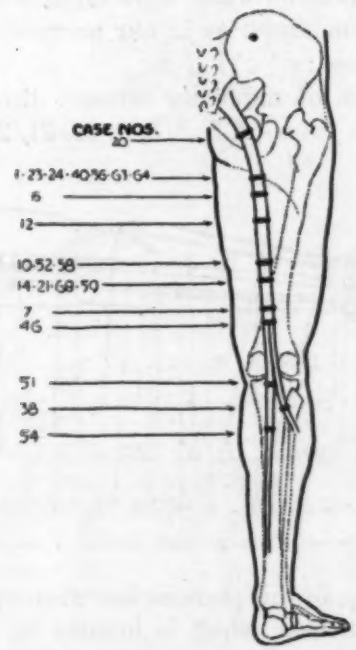

Fig. 3.—Levels of stimulation of the sciatic nerve and its branches.

plexus. A second type of internal nerve plexus must occur on account of the interfascicular rearrangement of different sensory functional groups of fibers as the functional arrangement in the spinal cord changes to that of various nerves.

When polysegmental nerves are brought to our attention, it becomes evident that fibers from two or more segments to one muscle or skin area (through some cutaneous nerve) must reach a fasciculus which goes to this muscle or skin area. This implies a third type of internal nerve plexus.

A more lengthy discussion of the many problems and a review of the literature will be postponed for a later paper.

PREPONDERANCE OF REACTION IN PROXIMAL MUSCLES

As a general rule it has been found that stimulation of the proximal part of the nerve does not cause contraction of muscles supplied by its distal portion. This rule has been found to apply to most nerves examined (see Table 1), and the individual case reports indicate this. Exceptions to the rule have occurred when mild currents were used, but the question of technic is apparently not the only factor since the exceptions were so rare. It seems probable that the contractions of the more powerful proximal muscles mask the actions of the smaller hand and foot muscles. Another factor may be the central localization of their fasciculi. Though we have no anatomic data to support the latter supposition, it seems probable that fasciculi for the most distal parts should in general be most centrally located.

STRAIGHT COURSE OF MOTOR FASCICULI

A review of the results for a given muscle at various levels brings out one point with certainty—*the course is a straight one from the point where the nerve has been made up by its contributing segments to the point of offset of the fasciculi as a branch.* An exception to this was uniformly found in one instance in which it was to be expected, namely, the fasciculi to the pronator radii teres. Further comment will be made in discussing the median nerve.

The straight course of fasciculi was commented on briefly by Marie in the case of the sciatic nerve. Stoffel's anatomic dissections implied it, but that is all. As far as we know, this point has not received the emphasis it deserves when its great importance for the knowledge of peripheral nerve anatomy is considered.

The reader may observe the basis of this statement by starting with a picture showing the results at the proximal end of a nerve and tracing the position of some particular bundle to the point of leaving the main trunk. Figures 40, 41, 42, 43, 45, 47, 48, 49, 51, 54, 55, 56 for example, will show the course of the fibers to the flexor carpi ulnaris.

Slight variations in position are no doubt due to slight rotation of the nerve trunk.

FINDINGS IN INDIVIDUAL NERVES AND THEIR BRANCHES

The nerves supplying the upper and lower extremities were stimulated at different points, from the vertebral foramina to the individual branches as they approached their muscles. The results of stimulation at the different levels were:

1. *Roots of the Brachial Plexus.*—One case, Case 41 (Figs. 4, 5 and 6).

The fifth and sixth cervical roots were not stimulated successfully.

Stimulation of the seventh cervical root gave contractions of the pectoral and triceps muscles.

Stimulation of the eighth cervical root at first caused contraction of the finger extensors, mainly of the extensor indicis; subsequent stimulations caused flexion of the fingers and thumb.

Figure 4

Figure 5

Fig. 4 (Case 41).—Normal brachial plexus, seventh cervical root; pectoralis major and triceps.

Fig. 5 (Case 41).—Normal brachial plexus, eighth cervical root; flexion of fingers and thumb.

Figure 6

Figure 7

Fig. 6 (Case 41).—Normal brachial plexus, first thoracic root; 1, flexion of fingers and wrist.

Fig. 7. (Case 5).—Abnormal brachial plexus, posterior cord: (1) triceps; (2) deltoid; (3) supinator longus.

The first thoracic root was associated with flexion of the fingers and wrist.

No observations were made on the primary trunks of the brachial plexus.

CORDS OF THE BRACHIAL PLEXUS: One case; Case 5 (Figs. 7 and 8).

The posterior cord of the brachial plexus was stimulated in one case resulting in contraction of the deltoid, triceps and supinator longus muscles. The lower cord was also stimulated in this case, resulting in flexion of the index and middle finger, on its inner aspect.

Figure 8

Figure 9

Fig. 8 (Case 5).—Abnormal brachial plexus, lower cord: (1) flexion of index and middle finger.

Fig. 9 (Case 31).—Normal median nerve, external head; pronator radii teres.

Figure 10

Figure 11

Fig. 10 (Case 31).—Abnormal median nerve, inner head: (1) flexor carpi radialis; (2) flexion of the fingers; (3) flexion of the thumb; (4) abductor pollicis.

Fig. 11 (Case 11).—Abnormal median nerve, inner head: (1) flexor carpi radialis; (2) palmaris longus; (3) flexor longus digitorum; (4) flexor longus pollicis.

INNER AND OUTER HEADS OF THE MEDIAN NERVE: The external head of the median nerve was stimulated in one case, *Case 31*[7] (Fig. 9), resulting in the contraction of the pronator radii teres over its entire circumference. Marie, Meige and Gosset also found this muscle represented in the external head.

7. Normal cases are italicized.

The inner head of the median nerve was stimulated twice; in both instances it was abnormal. In one case, Case 31 (Fig. 10), contraction resulted in the flexor carpi radialis, flexors of the fingers, thumb and the abductor pollicis. In another case, Case 11 (Fig. 11), the inner head of the median was abnormal and the result was contraction of the flexor carpi radialis, palmaris longus and flexors of the fingers and thumb. In neither instance was there a reaction in the pronator radii teres. Marie, Meige and Gosset found the flexors of the wrist and fingers in the inner head.

From the results of the stimulation of the roots, the different portions of the brachial plexus and the two heads of the median, certain conclusions may be drawn:

Flexion of the fingers results chiefly from the stimulation of the eighth cervical and first thoracic nerves.

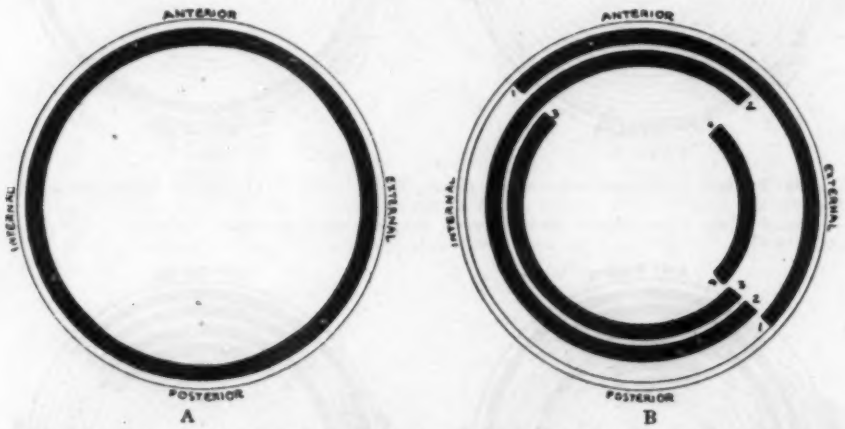

Fig. 12.—Results of stimulation of the two heads of the median nerve: *A*, external (from C6 and 7) pronator radii teres; *B*, internal (C8 and D1); (1) flexor carpi radialis; (2) flexors of the fingers; (3) flexion of the thumb; (4) abductor pollicis.

The triceps and pectoralis major react from the seventh cervical root and not from the eighth cervical and first thoracic. Sherren has observed slight contractions of these muscles on stimulating the sixth cervical root.

In stimulating the posterior cord, contraction occurs in the deltoid, triceps and supinator longus.

The external head of the median evidently supplies only the pronator radii teres muscle, indicating its origin from the sixth and seventh cervical segments (Fig. 12).

The inner head, receiving fasciculi from the eighth cervical and first thoracic roots, supplies all of the long forearm median muscles, in addition to the abductor pollicis, indicating that the supply of the last named muscle as given in textbooks as the sixth and seventh cervical is not entirely correct. It certainly receives fibers from the

eighth cervical and first thoracic. *This observation would explain the absence of involvement of the thumb muscles in the Erb type of brachial plexus paralysis.*

2. *Median.*—Eighteen cases; Cases 2 (Fig. 14); *3* (Fig. 20); 9 (Fig. 22), 13 (Fig. 19), 16 (Fig. 16), 17 (Figs. 34, 35 and 36), 19 (Figs. 26, 27 and 28), 26 (Fig. 21), 27 (Fig. 31, 32 and 33), 30 (Fig. 29), 36 (Fig. 17), 43 (Fig. 15), 44 (Fig. 18), 45 (Fig. 13), 50 (Fig. 24), 53 (Fig. 25), 67 (Fig. 23) and 69 (Fig. 30).

Figure 13

Figure 14

Fig. 13 (Case 45).—Abnormal median nerve: (1) pronator radii teres; (2) flexor profundus digitorum.

Fig. 14 (Case 2).—Abnormal median nerve: (1) pronator radii teres.

Figure 15

Figure 16

Fig. 15 (Case 43).—Abnormal median nerve: (1) pronator radii teres; (2) flexor carpi radialis; (3) flexion of the fingers; (4) flexor longus pollicis.

Fig. 16 (Case 16).—Abnormal median nerve: (1) pronator radii teres.

(*a*) Pronator Radii Teres: Fourteen cases; Cases 2 (Fig. 4), *3* (Fig. 20), 9 (Fig. 22), 13 (Fig. 19), 26 (Fig. 21), 31 (Fig. 10), 36 (Fig. 17), 43 (Fig. 15), 44 (Fig. 18), 45 (Fig. 13), 50 (Fig. 24), 53 (Fig. 25), 67(Fig. 23).

Figure 17

Figure 18

Fig. 17 (Case 36).—Abnormal median nerve: (1) pronator radii teres.

Fig. 18 (Case 44).—Abnormal median nerve: (1) pronator radii teres; (2) flexor sublimis digitorum; (3) flexor carpi radialis.

Figure 19

Figure 20

Fig. 19 (Case 13).—Abnormal median nerve: (1) pronator radii teres (greatest reaction antero-internally); (2) flexor carpi radialis; (3) palmaris longus; (4) flexion of the fingers.

Fig. 20 (Case 3).—Normal median nerve: (1) pronator radii teres; (2) flexor carpi radialis; (3) flexor longus pollicis.

Figure 21

Figure 22

Fig. 21 (Case 26).—Abnormal median nerve: (1) pronator radii teres (above lesion); (2) pronator radii teres (below lesion).

Fig. 22 (Case 9).—Abnormal median nerve: (1) flexion of wrist; (2) flexion of fingers; (3) flexion of thumb; (4) pronator radii teres.

Figure 23

Figure 24

Fig. 23 (Case 67).—Abnormal median nerve: (1) pronator radii teres; (2) flexor carpi radialis.

Fig. 24 (Case 50).—Abnormal median nerve: (1) pronator radii teres; (2) flexor carpi radialis; (3) flexor profundus digitorum.

Figure 25

Figure 26

Fig. 25 (Case 53).—Abnormal median nerve: (1) pronator radii teres; (2) flexor carpi radialis; (3) flexor profundus digitorum.

Fig. 26 (Case 19).—Abnormal median nerve: (1) sensory; (2) pronator quadratus; (3) opponens pollicis; (4) abductor pollicis; (5) lumbricales 1 and 2.

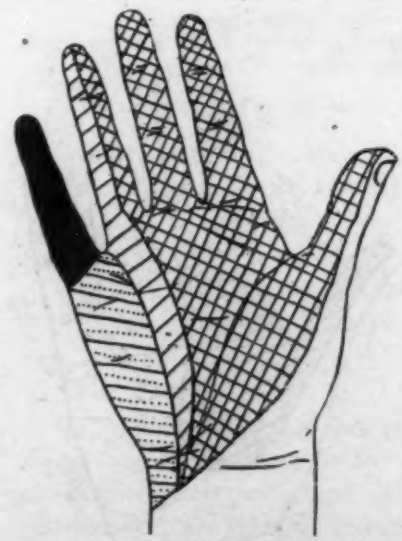

Figure 27

Figure 28

Fig. 27 (Case 19).—Black part denotes complete anesthesia; lines denote brush anesthesia; dots, pin-prick anesthesia (15 gm. of pressure).

Fig. 28 (Case 19).—Crossed lines denote hyperesthesia; black part, complete anesthesia; lines, brush anesthesia; dots, pin-prick anesthesia (15 gm. pressure).

Figure 29

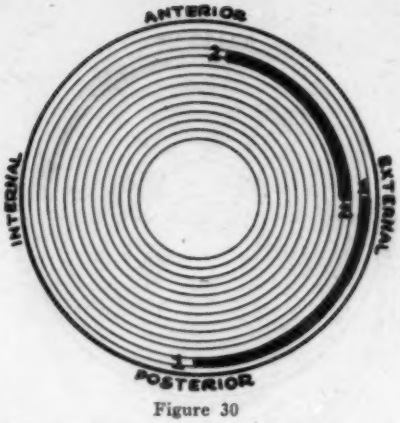

Figure 30

Fig. 29 (Case 30).—Abnormal median nerve: (1) flexor carpi radialis; (2) palmaris longus.

Fig. 30 (Case 69).—Abnormal median nerve: (1) pronator quadratus; (2) flexor longus pollicis.

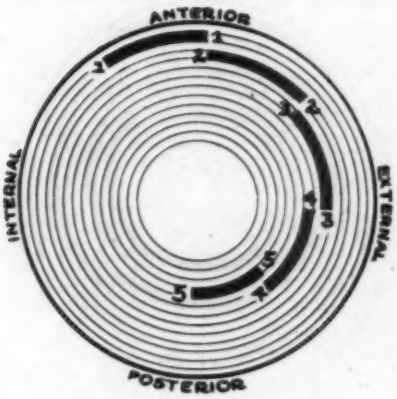

Figure 31

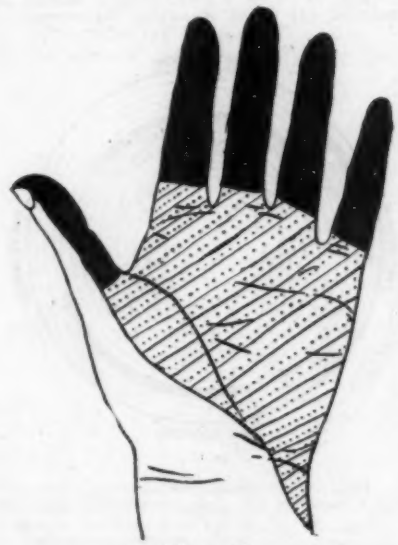

Figure 32

Fig. 31 (Case 27).—Abnormal median nerve; sensory findings—pain.

Fig. 32 (Case 27).—Black part denotes complete anesthesia; lines, brush anesthesia, dots, pin-prick anesthesia (15 gm. pressure).

Figure 33

Figure 34

Fig. 33 (Case 27).—Black part denotes complete anesthesia; lines, brush anesthesia, dots, pin-prick anesthesia (15 gm. pressure).

Fig. 34 (Case 17).—Lines denote brush anesthesia; dots, pin-prick anesthesia (15 gm. pressure).

This muscle has been stimulated from the external head of the median to the point at which the branch goes to the muscle. The branches come from the external head of the median (cervical 6 and 7) and leave the nerve internally. They must therefore cross the nerve as they descend. One of these cases, stimulated above the upper third arm, showed this fasciculus on the external aspect. From here to the elbow, two cases show a response on the anterior surface of the nerve. In one case (Case 26, Fig. 21) the muscle responded both externally and internally, the former when stimulated rather high above the lesion, the latter when stimulated low, below the lesion. Anatomic dissection has borne out these observations, and the fibers are found passing from the external to internal part on the anterior

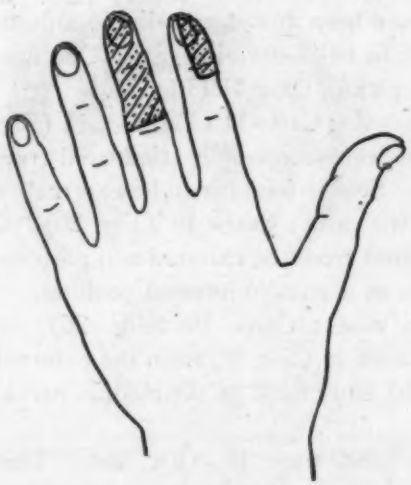

Figure 35

Figure 36

Fig. 35 (Case 17).—Lines denote brush anesthesia; dots, pin-prick anesthesia (15 gm. pressure).

Fig. 36 (Case 17).—Abnormal median nerve: (1) sensory findings—pain.

surface of the nerve. Marie, Meige and Gosset found both pronators antero-external. Stoffel found the pronator radii teres on the external surface. He has shown the crossing which we have confirmed, in one of the figures in his article.

(*b*) Flexor Carpi Radialis: Twelve cases; Cases *3* (Fig. 20), 9 (Fig. 22), 11 (Fig. 1), 13 (Fig. 19), 16 (Fig. 16), 30 (Fig. 29, 31 Fig. 10), 43 (Fig. 15), 44 (Fig. 18), 50 (Fig. 24), 53 (Fig. 25), 67 (Fig. 23).

The fibers supplying this muscle have been found occupying an antero-internal position in the trunk of the median nerve. Stoffel places them externally, and Marie, Meige and Gosset are uncertain whether they are internal or external.

(*c*) Flexor Sublimis and Flexor Profundus Digitorum: Eleven cases: Cases 5 (Fig. 8), 9 (Fig. 22), 11 (Fig. 11), 13 (Fig. 19), 31 (Fig. 10), 41 (Figs. 5 and 6), 43 (Fig. 15), 44 (Fig. 18), 45 (Fig. 13), 50 (Fig. 24), 53 (Fig. 25).

These muscles have been considered together as it is impossible to distinguish the difference in their action under the conditions existing during a surgical operation. The position of fibers to these muscles has been found to be antero-internal. Marie, Meige and Gosset found these postero-internally. Stoffel puts the profundus postero-internally, but the sublimis internally. Two cases were stimulated through the brachial plexus. (Case 41, Figs. 5 and 6; Case 5, Fig. 8).

(*d*) Flexor Longus Pollicis: Seven cases; Case *3* (Fig. 20), 9 (Fig. 22), 11 (Fig. 11), 31 (Fig. 10), 41 (Fig. 6), 43 (Fig. 15), 69 (Fig. 30), These fibers have been found anterior to antero-internal. Stoffel found these fibers to be postero-internal. One case was stimulated through the brachial plexus, Case 41 (Fig. 5).

(*e*) Palmaris Longus: Three cases; Cases 11 (Fig. 11), 13 (Fig. 19), 30 (Fig. 29). This muscle has been successfully stimulated from the anterior surface of the median. Stoffel found it to be external.

(*f*) Pronator Quadratus: Two cases; Cases 19 (Fig. 26), 69 (Fig. 30). This muscle was stimulated from the external and postero-external aspects. Stoffel places this in a postero-internal position.

(*g*) Abductor Pollicis: Two cases; Cases 19 (Fig. 26), 31 (Fig. 10). This muscle was stimulated, in Case 19, from the external aspect of the nerve and also from the inner head of the median nerve, external aspect, in Case 31.

(*h*) Opponens Pollicis: One case, Case 19, (Fig. 26). This muscle was stimulated from the postero-internal aspect. Stoffel places all the thenar muscles anteriorly, occupying three fifths of the nerve. Marie, Meige and Gosset place them posteriorly.

(*i*) First and Second Lumbricales: One case, Case 19 (Fig. 26). These muscles were stimulated from the posterior-external aspect of the nerve.

In view of the fact that the action of the lumbricales muscles has been the subject of some discussion, it may be stated that the result of their stimulation in this case, *was flexion of the metacarpophalangeal joints of the first and second fingers.*

Déjerine and Mouzon have made no observations on the nerve. Their findings on other nerves will be quoted hereafter.

Sensory: Three cases, Cases 17 (Fig. 36), 19 (Fig. 26), 27 (Fig. 31). Three patients were operated on under local anesthesia. In two cases, sensory reactions of pain were caused from stimulation of the anterior aspect, the patients stating "it hurts," "it burns." In Case 27,

pain was localized at the base of the index, middle, ring and little fingers as the electrode was passed from the posterior aspect around the external to the anterior surface. No reaction of pain resulted from stimulation of the internal surface of the nerve. Stoffel places the sensory bundle anteriorly.

The foregoing observations on the median nerve brings out these points:

1. The bundles of the long forearm muscles lie anteriorly and internally in the trunk of the median nerve (Fig. 37).

2. The bundles of the intrinsic hand muscles are in the main posterior (Fig. 38).

Figure 37

Figure 38

Fig. 37.—Results of stimulation of the median nerve at the midarm level: (1) pronator radii teres; (2) flexor carpi radialis; (3) palmaris longus; (4) flexores digitorum; (5) flexor pollicis; (6) pronator quadratus.

Fig. 38.—Results of stimulation of the median nerve at midforearm level: (1) sensory; (2) opponens pollicis; (3) abductor pollicis; (4) lumbricales.

3. Sensory fibers (pain) are in the main anterior (Fig. 38),

3. *The Ulnar Nerve*: Nineteen cases, Cases 3 (Fig. 46), 8 (Fig. 51), 11 (Fig. 40), 13 (Fig. 44, 16 (Fig. 41), *18* (Fig. 54), 27 (Fig. 57), 28 (Fig. 50), 31 (Fig. 39), 34 (Fig. 55), 35 (Fig. 56), 42 (Fig. 49), 43 (Fig. 43), 45 (Fig. 42), 47 (Fig. 52), 49 (Fig. 53), 50 (Fig. 47), 60 (Fig. 45), 66 (Fig. 48).

This nerve is readily stimulated. Frequently it may be investigated in the normal condition due to its proximity to the median. The results of stimulation have been very satisfactory.

(*a*) Flexor Carpi Ulnaris: Fifteen cases; Cases 3 (Fig. 46), 8 (Fig. 51), 11 (Fig. 40), 16 (Fig. 41), *18* (Fig. 54), 31 (Fig. 39), 34 (Fig. 55), 35 (Fig. 56), 41 (Fig. 6), 42 (Fig. 49), 43 (Fig. 43), 45 (Fig. 42), 50 (Fig. 47), 60 (Fig. 45), 66 (Fig. 48). The position of its fibers is on the internal aspect of the nerve. Marie, Meige and Gosset and Stoffel also find the fibers internal. Déjerine and Mouzon find that they are external.

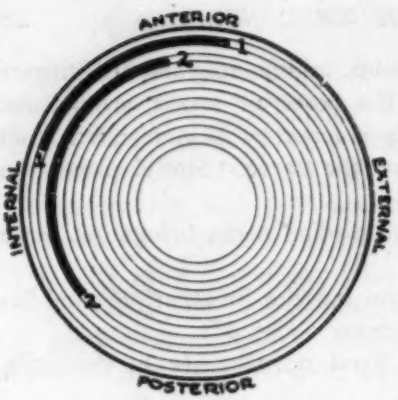

Figure 39

Figure 40

Fig. 39 (Case 31).—Abnormal ulnar nerve: (1) flexor carpi ulnaris; (2) flexor profundus digitorum.

Fig. 40 (Case 11).—Abnormal ulnar nerve: (1) flexor carpi ulnaris; (2) flexor profundus digitorum.

Figure 41

Figure 42

Fig. 41 (Case 16).—Abnormal ulnar nerve: (1) hypothenar muscles; (2) interossei; (3) adductor pollicis; (4) flexor carpi ulnaris; (5) flexor profundus digitorum.

Fig. 42 (Case 45).—Abnormal ulnar nerve: (1) flexor carpi ulnaris; (2) flexor profundus digitorum.

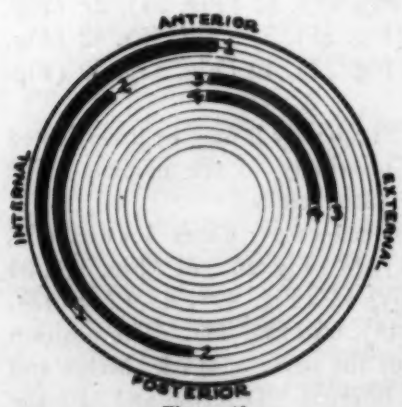

Figure 43

Figure 44

Fig. 43 (Case 43).—Abnormal ulnar nerve: (1) flexor carpi ulnaris; (2) flexor profundus digitorum; (3) adductor pollicis; (4) hypothenar muscles.

Fig. 44 (Case 13).—Abnormal ulnar nerve: (1) flexor profundus digitorum.

Figure 45

Figure 46

Fig. 45 (Case 60).—Abnormal ulnar nerve: (1) flexor carpi ulnaris.
Fig. 46 (Case 3).—Abnormal ulnar nerve: (1) flexor carpi ulnaris; (2) flexor profundus digitorum.

Figure 47

Figure 48

Fig. 47 (Case 50).—Abnormal ulnar nerve: (1) flexor carpi ulnaris.
Fig. 48 (Case 66).—Abnormal ulnar nerve: (1) flexor carpi ulnaris; (2) flexor profundus digitorum; (3) interossei; (4) adductor pollicis; (5) abductor minimi digiti.

Figure 49

Figure 50

Fig. 49 (Case 42).—Abnormal ulnar nerve: (1) flexor caspi ulnaris; (2) flexor profundus digitorum.
Fig. 50 (Case 28).—Abnormal ulnar nerve: (1) interossei; (2) adductor pollicis.

Figure 51

Figure 52

Fig. 51 (Case 8).—Abnormal ulnar nerve: (1) flexor carpi ulnaris; (2) flexor profundus digitorum.
Fig. 52 (Case 47).—Abnormal ulnar nerve: (1) interossei.

Figure 53

Figure 54

Fig. 53 (Case 49).—Abnormal ulnar nerve: (1) intrinsic muscles of the hand.
Fig. 54 (Case 18).—Normal ulnar nerve: (1) adductor pollicis; (2) flexor carpi ulnaris; (3) flexor profundus digitorum.

Figure 55

Figure 56

Fig. 55 (Case 34).—Abnormal ulnar nerve: (1) flexor carpi ulnaris; (2) flexor profundus digitorum.
Fig. 56 (Case 35).—Abnormal ulnar nerve: (1) flexor carpi ulnaris.

(*b*) FLEXOR PROFUNDUS DIGITORUM: Thirteen cases; Cases 3 (Fig. 46), 8 (Fig. 51), 11 (Fig. 40), 13 (Fig. 44), 16 (Fig. 41), *18* (Fig. 54), 31 (Fig. 39), 34 (Fig. 55), 41 (Fig. 6), 42 (Fig. 49), 43 (Fig. 44), 45 (Fig. 43), 66 (Fig. 48). The fibers of this muscle were found internally, either posterior to those of the flexor carpi ulnaris or in the same position. Marie, Meige and Gosset found that the fibers were postero-external, Déjerine and Mouzon external, and Stoffel internal and posterior to the flexor carpi ulnaris.

(*c*) ADDUCTOR POLLICIS: Six cases; Cases 16 (Fig. 41), *18* (Fig. 54), 28 (Fig. 50), 43 (Fig. 43), 49 (Fig. 53), 66 (Fig. 48).

The fibers of this muscle lie antero-externally. Déjerine and Mouzon place them postero-externally and Stoffel places them posteriorly.

(d) INTEROSSEI: Five cases; Cases 16 (Fig. 41), 28 (Fig. 50), 47 (Fig. 52), 49 (Fig. 53) 66 (Fig. 48).

Figure 57

Figure 58

Fig. 57 (Case 27).—Abnormal ulnar nerve, sensory findings in ulnar distribution—pain: (1) palmar surface; (2) ulnar surface; (3) dorsal surface.
Fig. 58.—Results of stimulation of the ulnar nerve: (1) flexor carpi ulnaris; (2) flexor profundus digitorum; (3) hypothenars; (4) interossei; (5) adductor pollicis; (6) sensory.

These fibers are antero-external in all but Case 47, in which these were the only muscles reacting, the rest of the nerve being diseased. This probably accounts for the discrepancy. Déjerine and Mouzon find the fibers for these muscles are internal, Stoffel posterior.

(*e*) HYPOTHENARS: Four cases; Cases 16 (Fig. 41) 43 (Fig. 43), 49 (Fig. 52), 66 (Fig. 48).

These muscles are considered together as it is difficult to obtain sufficiently clear reactions to differentiate their action with certainty. Their fibers are situated antero-externally in the nerve. Déjerine and Mouzon place them internally, Stoffel posteriorly. These muscles were, according to Marie, Meige and Gosset, of uncertain localization but on clinical grounds were placed on the internal and anterior aspect of the nerve.

SENSORY: One case; Case 27 (Fig. 57).

Only one case was done under local anesthesia. In this the result was clear cut. On stimulating the nerve antero-internally, pain was felt in the palmar distribution of the ulnar; stimulation antero-externally caused pain on the ulnar aspect of the hand; and postero-externally on the dorsal aspect in the ulnar distribution. As far as we can find, these observations on the location of sensory fibers in the ulnar and median nerves (2) are the first to be recorded. Figure 58 shows the composite results of the ulnar cases.

4. *Circumflex Nerve.*—Three cases; Cases 5 (Fig. 7), *31* (Fig. 59), 39 (Fig. 60).

Figure 59

Figure 60

Fig. 59 (Case 31).—Abnormal circumflex nerve: (1) deltoid.
Fig. 60 (Case 39).—Abnormal circumflex nerve: (1) deltoid.

Case 39 showed a reaction in the deltoid on all aspects of the nerve. In Case 31 the deltoid was also stimulated through the brachial plexus. In Case 5 this muscle reacted on the anterior aspect of the posterior cord.

5. *Musculo-Spiral Nerve.*—Four cases; Cases 15 (Fig. 63), 22 (Fig. 64), 31 (Fig. 61), 33 (Fig. 62).

The number of cases of musculo-spiral injury in which a reaction could be obtained was small, the nerve being divided anatomically or physiologically in most instances. Four cases yielded results.

(*a*) TRICEPS: Four cases; Cases 5 (Fig. 7), 31 (Fig. 61), 33 (Fig. 62), 41 (Fig. 4).

The position of this muscle is easily determined. It is internal, running somewhat posteriorly. The bundle is readily seen. The triceps were also stimulated through the brachial plexus in two cases, Cases 5 (Fig. 7), 41 (Fig. 4).

(*b*) SUPINATOR LONGUS: Two cases; Case 5 (Fig. 7), Case 31 (Fig. 61).

This muscle reacted in one case and its fibers lay externally. Marie, Meige and Gosset, Déjerine and Mouzon and Stoffel all agree with us. Case 5 was stimulated through the brachial plexus.

(c) Extensor Carpi Radialis: Two cases, Cases 31 (Fig. 61), 22 (Fig. 64).

These muscles were found anteriorly in Case 31, posteriorly in Case 22. The discrepancy is probably due to torsion of the nerve as it passes around the humerus. The level of Case 31 was in the axilla, that of Case 22, below the elbow. Marie, Meige and Gosset place these fibers internally; Déjerine and Mouzon and Stoffel postero-externally.

(d) Extensors of the Fingers and Thumb: One case, Case 15 (Fig. 63).

In this case, the extensor indicis reacted on the internal side of the nerve. Marie, Meige and Gosset, Déjerine and Mouzon, and Stoffel found these fibers to be postero-internal.

6. *Sciatic Nerve.*—Twenty-two cases; Cases 1 (Fig. 75), 6 (Fig. 79), *7* (Fig. 85), 10 (Figs. 80, 81, 89), 12 (Fig. 70), 14 (Figs. 71, 90), *20* (Fig. 66), 21 (Fig. 72), 23 (Fig. 67), 24 (Fig. 68), 38 (Fig. 92), *40* (Fig. 76), 46 (Fig. 91), 51 (Fig. 86), 52 (Fig. 82), 54 (Fig. 87), *56* (Fig. 77), 58 (Fig. 83), 59 (Figs. 73, 93), 63 (Fig. 78), *64* Fig. 69), 68 (Fig. 84).

This nerve was stimulated at various levels.

Sciatic Trunk: Eight cases; Cases 12 (Fig. 70), 14 (Fig. 71), *20* (Fig. 66), 21 (Fig. 72), 23 (Fig. 69), 24 (Fig. 68), 59 (Fig. 73), *64* (Fig. 69).

1. *Internal Popliteal Division.*[8]— Five cases; Cases 12 (Fig. 70), *20* (Fig. 66), 23 (Fig. 67), 24 (Fig. 68), *64* (Fig. 69).

(a) Hamstring Muscles: Three cases; Cases *20* (Fig. 66), 23 (Fig. 67), *64* (Fig. 69).

The fibers lie postero-internally.

(b) Gastrocnemius and Soleus Croup: Four cases; Cases 12 (Fig. 70), *20* (Fig. 66), 24 (Fig. 68), *64* (Fig. 69).

The fibers were uniformly found on the posterior side internal to the midline between the internal and external division of the nerve.

(c) Tibialis Posticus: Three cases; Cases 12 (Fig. 70), *20* (Fig. 66), *64* (Fig. 69).

In all three cases fibers to this muscle were found anteriorly and internal to the midline of the trunk.

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8. The use of the word "division" indicates that the two nerves had not been separated and that the trunk was stimulated as a whole.

Figure 61

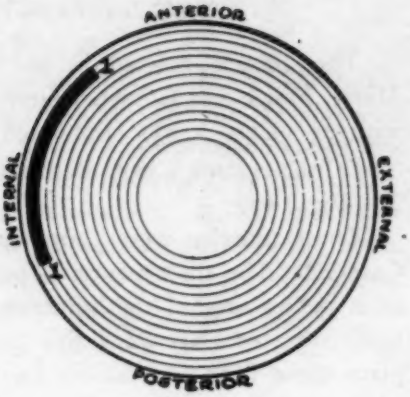

Figure 62

Fig. 61 (Case 31).—Abnormal musculospiral nerve: (1) triceps; (2) supinator longus; (3) extensor carpi radialis.
Fig. 62 (Case 33).—Abnormal musculospiral nerve: (1) triceps.

Figure 63

Figure 64

Fig. 63 (Case 15).—Abnormal musculospiral nerve: (1) extensor indicis.
Fig. 64 (Case 22).—Abnormal musculospiral nerve: (1) extensor carpi radialis; greatest reaction posteriorly.

Figure 65

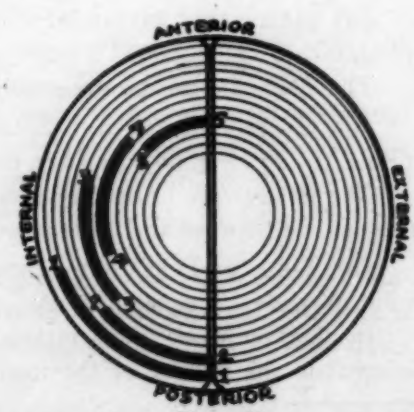

Figure 66

Fig. 65.—Results of stimulation of the musculospiral nerve: (1) triceps; (2) supinator longus; (3) extensor carpi radialis.
Fig. 66 (Case 20).—Normal sciatic nerve: (1) ham strings; (2) gastrocnemius; (3) flexor longus hallucis; (4) flexor longus digitorum; (5) tibialis posticus.

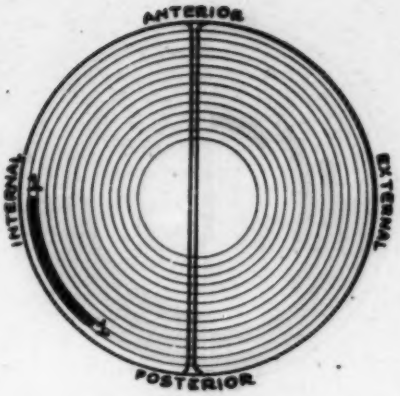

Figure 67

Figure 68

Fig. 67 (Case 23).—Abnormal sciatic nerve: (1) hamstrings.
Fig. 68 (Case 24).—Abnormal sciatic nerve: (1) gastrocnemius and soleus.

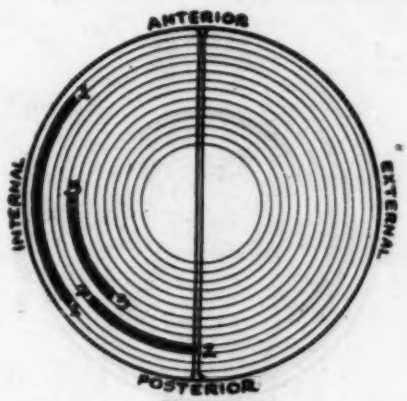

Figure 69

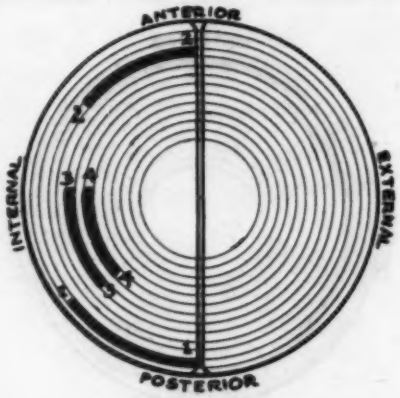

Figure 70

Fig. 69 (Case 64).—Abnormal sciatic nerve: (1) biceps; (2) gastrocnemius; (3) tibialis posticus.
Fig. 70 (Case 12).—Abnormal sciatic nerve: (1) gastrocnemius; (2) tibialis posticus; (3) flexor longus digitorum; (4) flexor longus hallucis.

Figure 71

Figure 72

Fig. 71 (Case 14).—Abnormal sciatic nerve: (1) peronei; (2) tibialis anticus; (3) both extensors of the toes.
Fig. 72 (Case 21).—Abnormal sciatic nerve: (1) peronei.

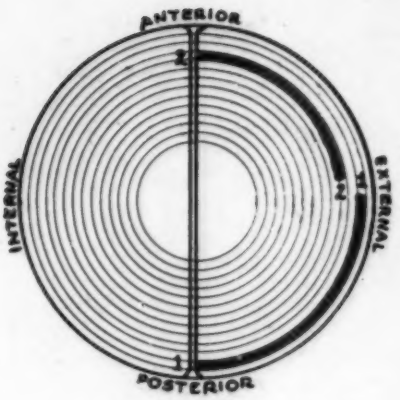

Figure 73

Figure 74

Fig. 73 (Case 59).—Abnormal sciatic nerve: (1) peronei; (2) tibialis anticus.
Fig. 74.—Results of stimulation of the sciatic nerve: (1) hamstrings; (2) gastrocnemius and soleus; (3) flexor longus hallucis; (4) flexor longus digitorum; (5) tibialis posticus; (6) peronei; (7) tibialis anticus; (8) extensors of the toes.

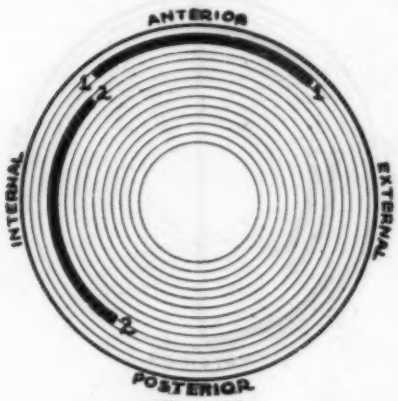

Figure 75

Figure 76

Fig. 75 (Case 1).—Abnormal internal popliteal nerve: (1) tibialis posticus; (2) gastrocnemius and soleus.
Fig. 76 (Case 40).—Normal internal popliteal nerve: (1) gastrocnemius and soleus; (2) tibialis posticus; (3) flexor longus digitorum; (4) flexor longus hallucis.

Figure 77

Figure 78

Fig. 77 (Case 56).—Normal internal popliteal nerve: (1) gastrocnemius and soleus; (2) tibialis posticus.
Fig. 78 (Case 63).—Abnormal internal popliteal nerve: (1) biceps femoris; (2) gastrocnemius; (3) tibialis posticus.

Figure 79

Figure 80

Fig. 79 (Case 6).—Abnormal internal popliteal nerve: (1) tibialis posticus; (2) flexor longus hallucis; (3) flexor longus digitorum.

Fig. 80 (Case 10).—Abnormal internal popliteal nerve: (1) gastrocnemius and soleus; (2) tibialis posticus; (3) flexor longus digitorum; (4) flexor longus hallucis.

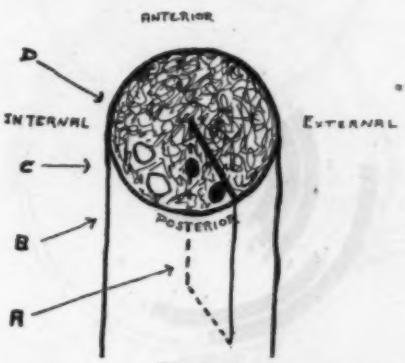

Figure 81

Figure 82

Fig. 81 (Case 10).—*A*, plane of incision in internal popliteal nerve; *B*, the black areas indicate bundles which were normal; *C*, white areas indicate bundles which were abnormal; *D*, interstitial and scar tissue.

Fig. 82 (Case 52).—Abnormal internal popliteal nerve: (1) gastrocnemius and soleus.

Figure 83

Figure 84

Fig. 83 (Case 58).—Abnormal internal popliteal nerve: (1) Gastrocnemius and soleus; (2) tibialis posticus.

Fig. 84 (Case 68).—Abnormal internal popliteal nerve: (1) gastrocnemius and soleus; (2) tibialis posticus; (3) intrinsic foot muscles.

Figure 85

Figure 86

Fig. 85 (Case 7).—Normal internal popliteal nerve: (1) gastrocnemius and soleus; (2) tibialis posticus; (3) flexor longus hallucis; (4) flexor longus digitorum.

Fig. 86 (Case 51).—Abnormal internal popliteal nerve: (1) plantar muscles; (2) flexor minimi digiti; (3) flexor brevis digitorum.

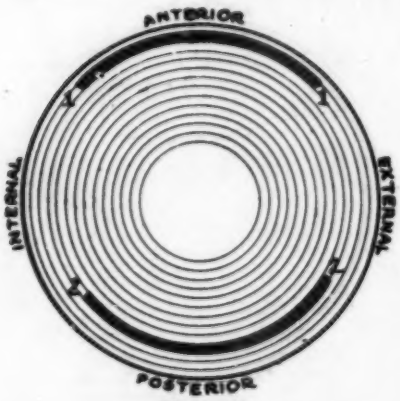

Figure 87

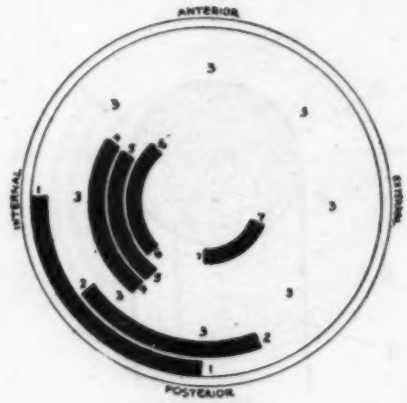

Figure 88

Fig. 87 (Case 54).—Abnormal internal popliteal nerve: (1) tibialis posticus; (2) flexor longus digitorum.

Fig. 88.—Results of stimulation of the internal popliteal nerve: (1) hamstrings; (2) gastrocnemius; (3) tibialis posticus; (4) flexor longus digitorum; (5) flexor longus hallucis; (6) intrinsic; (7) flexor brevis hallucis.

Figure 89

Figure 90

Fig. 89 (Case 10).—Abnormal external popliteal nerve: (1) peronei.

Fig. 90 (Case 14).—Abnormal external popliteal nerve: (1) peronei; (2) tibialis anticus; (3) extensors of the toes.

(d) FLEXOR LONGUS DIGITORUM: Two cases; Cases 12 (Fig. 70), *20* (Fig. 66).

In these cases the fibers lie postero-internally.

(e) FLEXOR LONGUS HALLUCIS: Two cases; Cases 12 (Fig. 70), *20* (Fig. 66).

Fibers to this muscle were found in the same position as those to the flexor longus digitorum. Reaction of the intrinsic muscles of the

Figure 91

Figure 92

Fig. 91 (Case 46).—Abnormal external popliteal nerve: (1) peronei.
Fig. 92 (Case 38).—Abnormal external popliteal nerve: (1) peronei.

Figure 93

Fig. 93 (Case 59).—Abnormal external popliteal nerve: (1) peronei; (2) tibialis anticus.

foot was not obtained in any of the cases in which the trunk was stimulated.

2. *External Popliteal Division.*—Three cases; Cases 14 (Fig. 71), 21 (Fig. 72), 59 (Fig. 73).

(*a*) PERONEI: Three cases; Cases 14 (Fig. 71), 21 (Fig. 72), 59 (Fig. 73).

These muscles lie posteriorly, external to the midline dividing the trunk into its divisions.

(*b*) TIBIALIS ANTICUS: Two cases; Cases 14 (Fig. 71), 59 (Fig. 73).

This muscle was stimulated twice from the trunk of the sciatic; its fibers lay in the same position as those of the peronei, i. e., postero-externa, in Case 14, and anterio-external in Case 59.

(*c*) EXTENSORS OF THE TOES AND GREAT TOE: One case; Case 14 (Fig. 71).

The position was the same as for the peronei. Figure 74 shows the composite results of stimulations of the sciatic nerve.

7. Internal Popliteal Nerve.—Twelve cases; Cases 1 (Fig. 75), 6 (Fig. 79), *7* (Fig. 85), 10 (Fig. 80), *40* (Fig. 76), 51 (Fig. 86), 52 (Fig. 82), 54 (Fig. 87), *56* (Fig. 77), 58 (Fig. 83), 63 (Fig. 78), 68 (Fig. 84).

(*a*) HAMSTRING MUSCLES: One case; Case 63 (Fig. 78).

The position is internal. This position corresponds to that found in four cases stimulated in the trunk. Stoffel also gives it as internal.

(b) GASTROCNEMIUS AND SOLEUS: Nine cases; Cases 1 (Fig. 75), *7* (Fig. 85), 10 (Fig. 80), *40* (Fig. 76), 52 (Fig. 82), *56* (Fig. 77), 58 (Fig. 83), 63 (Fig. 78), 68 (Fig. 84).

In Case 1, the fibers reacted anteriorly. In Cases *7*, 10, *40*, *56*, 58, 63 and 68, they reacted posteriorly; in case 52, on the internal side from the anterior to the posterior poles. With one exception, therefore, and this was the first case tried, the fibers were located in the same position, posteriorly. Marie, Meige and Gosset, and Stoffel found them posteriorly, Déjerine and Mouzon placed them internally.

(*c*) TIBIALIS POSTICUS: Ten cases; Cases 1 (Fig. 75), 6 (Fig 79), *7* (Fig. 85), 10 (Fig. 80), *40* (Fig. 78), 54 (Fig. 87), *56* (Fig. 77), 58 (Fig. 83), 63 (Fig. 78), 68 (Fig. 84).

The position in which this muscle could be stimulated varied considerably. We have no adequate explanation for this.

Cases 1 (Fig. 75), *7* (Fig. 85) internal reaction. Cases 10 (Fig. 80), *40* (Fig. 76), 54 (Fig. 87) anterior reaction. Case 6 (Fig. 79) posterior reaction. Cases *56* (Fig. 77), 58 (Fig. 83), *64* (Fig. 69) postero-internal reaction. Case 63 (Fig. 78) postero-external reaction. Case 68 (Fig. 84) antero-external reaction.

Marie, Meige and Gosset found that these fibers were antero-internal; Stoffel, antero-external; and Déjerine and Mouzon, external.

(d) FLEXOR LONGUS DIGITORUM: Five cases; Cases 6 (Fig. 80), *7* (Fig. 85), 10 (Fig. 80), *40* (Fig. 76), 54 (Fig. 87).

Cases 6, 10 and *40* reacted internally, Case *7*, antero-internally and Case 54 posteriorly. Therefore, in four cases the position was internal or antero-internal and in one, posterior. The last position appears to be incorrect in view of the isolation and the position of the next

muscle. The fasciculi of these two muscles would reasonably lie together. Marie, Meige and Gosset give this as postero-internal, Déjerine and Mouzon as external and Stoffel as antero-internal.

(e) FLEXOR LONGUS HALLUCIS: Four cases; Cases 6 (Fig. 80), *7* (Fig. 85), 10 (Fig. 80), *40* (Fig. 76).

This muscle was found internally or postero-internally in each case. Marie, Meige and Gosset found its position to be postero-internal, and Déjerine, Mouzon and Stoffel found it external.

(*f*) INTRINSIC MUSCLES OF THE FOOT: Two cases; Cases 51 (Fig. 87), 68 (Fig. 84).

Fibers to the plantar muscles, interossei and muscles of the small toe lay internally, while those to the flexor brevis digitorum lay postero-externally. Figure 88 shows results of stimulation of internal popliteal nerve.

8. *External Popliteal Nerve.*—Five cases; Cases 10 (Fig. 89), 14 (Fig. 90), 38 (Fig. 92), 46 (Fig. 91), 56 (Fig. 93).

Figure 94

Fig. 94.—Results of stimulation of the external popliteal nerve: (1) peronei; (2) tibialis anticus; (3) extensors of the toes.

(*a*) PERONEI: Five cases; Cases 10 (Fig. 89), 14 (Fig. 90), 38 (Fig. 92), 46 (Fig. 91), 59 (Fig. 93).

The fibers were found postero-internally in Cases 10, 14 and 38. In Case 46, in which the nerve was greatly damaged and but four or five bundles remained, the position was postero-external. The results obtained in the trunks agree with these. The apparent difference between the position is explained by the splitting of the sciatic trunk. Thus this group of fibers assumes a postero-internal position in the external popliteal nerve and a postero-external position in the trunk. Marie, Meige and Gosset placed them in a posterior position, Déjerine and Mouzon in an external position, and Stoffel in a postero-internal position.

(*b*) TIBIALIS ANTICUS: Two cases; Cases 14 (Fig. 90), 59 (Fig. 93).

These fibers lay externally. Marie, Meige and Gosset found their position antero-internal, Déjerine and Mouzon, internal and Stoffel, external.

(*c*) EXTENSORS OF THE TOES: One case; Case 14 (Fig. 90).

These fibers lay externally. Marie, Meige and Gosset placed them antero-externally, Déjerine and Mouzon, internally and Stoffel externally. Figure 94 shows composite results of stimulation of the external popliteal nerve.

REPORT OF CASES

For purposes of brevity the following abbreviations are used in the case reports presented:

Lieutenant-Colonel Frazier, F.
Major Coleman, C.
Abductor pollicis, Ab. Pol.
Adductor pollicis, Ad. Pol.
Biceps, Bi.
Deltoid, Del.
Faradic, F.
Flexor brevis hallucis, F. B. H.
Flexor carpi radialis, F. C. R.
Flexor carpi ulnaris, F. C. U.
Flexor longus digitorum, F. L. D.
Flexor longus hallucis, F. L. H.
Flexor longus pollicis, F. L. P.
Flexor profundus digitorum, F. P. D.
Flexor sublimis digitorum, F. S. D.
Galvanic, G.
Gastrocnemius, Gast.
Hamstrings, Ham.
Major King, Kg.
Captain Knapp, Kp.
Hypothenar, Hr.
Interossei, Io.
Lumbricales, Lum.
Operation, Op.
Palmaris longus, P. L.
Pectoralis major, P. M.
Peronei, Per.
Pronator quadratus, P. Q.
Pronator radii teres, P. R. T.
Soleus, Sol.
Supinator longus, S. L.
Tibialis anticus, T. A.
Tibialis posticus, T. P.
Triceps, Tri.
Voluntary movement, V. M.
Wounded, Wd.

CASE 41.[9]—G. B. (Figs. 4, 5, 6); wd. July 16, 1918; op. June 23, 1919, F. Diag.: Complete interruption of the fifth and sixth roots of the brachial plexus. Nerves Stimulated: Seventh and eighth cervical and first dorsal roots. Muscles Reacting: P. M., Tri. and flexors of the wrist, fingers and thumb. V. M.: Weak external rotation of the arm: no flexion of the forearm; otherwise normal.

CASE 5.—A. C. (Figs. 7, 8); wd. Sept. 29, 1918; op. May 13, 1919, F. Diag.: Partial paralysis of the left brachial plexus, the cords of which were embedded in scar tissue 4 cm. below the clavicle. Nerves Stimulated: Posterior and lower cords of the brachial plexus. Muscles Reacting: Tri., Del., S. L., through the posterior cord, and the flexors of the index and middle fingers through the lower cord. Elec. Reaction (before op.): F.; none in the affected muscles. G.: slow contractions without polar reversal. V. M.: Flexion of the fingers and adduction of the thumb were the only movements observed.

Note.—This is an example of the phenomenon of difference between direct and percutaneous stimulation, in that the muscles which reacted at operation failed to do so at the previous clinical examination.

9. Sequence of cases as on Figures 2 and 3.

Case 31.—M. P. A. (Figs. 9, 10, 39, 59, 61); wd. July 18, 1918; op. June 5, 1919, F. Diag.: Lesion of the right musculospiral, ulnar and lower head of the median nerves, with neuromas of the musculospiral and ulnar. Nerves Stimulated and Muscles Reacting: circumflex, deltoid, musculospiral, Tri., S. L. and E. C. R.; median (outer head) P. R. T. (inner head), F. C. R., F. P. D., Ab. Pol.; ulnar, F. C. U., F. P. D. Elec. Reac.: F.: Reactions present in Tri., P. R. T., F. C. R., F. S. D., F. P. D. None in S. L., E. C. R. or F. C. U. G.: Slow reactions without reversal in all of the above muscles. V. M.: Weak in Opp. and Abd. P., absent in other affected muscles.

Note.—In this case the phenomenon of difference was obtained in several muscles: S. L., E. C. R. and F. C. U. The level of stimulation is not shown in the table, but was about the same as in Case 11.

Case 11.—H. J. M. (Figs. 11, 40); wd. Sept. 12, 1918; op. May. 21, 1919, F. Diag.: Lesion of the left median, ulnar and lesser internal cutaneous nerves; outer head of median involved in scar tissue. Nerves Stimulated and Muscles Reacting: ulnar: F. C. U., F. P. D.; median (inner head): F. C. R., P. L., F. L. D. and F. L. P.

Case 45.—H. V. (Figs. 13, 42); wd. Sept. 27, 1918; op July 23, 1919, C. Diag.: Partial paralysis of the median and ulnar nerves; neuromata. Nerves Stimulated and Muscles Reacting: ulnar: F. C. U., F. P. D.; median: P. R. T., F. P. D.

Case 2.—E. E. (Fig. 14); wd. July 18, 1918; op. May 5, 1919, F. Diag.; Complete lesions of the musculospiral and median nerves. Nerve Stimulated: median. Muscle Reacting: P. R. T. Elec. Reac. F.: none. G.: slow contraction with polar reversal in median muscles. V. M.: none.

Note.—Phenomenon of difference was obtained in this case in the P. R. T.

Case 43.—W. S. (Figs. 15, 43); wd. July 31, 1918; op. June 29, 1919, C. Diag.: Partial lesion of the median and ulnar nerves. Lateral neuroma of the median and causalgia. Nerves Stimulated and Muscles Reacting: median, P. R. T., F. C. R., F. P. D. and F. L. P.; ulnar, F. C. U., F. P. D., Ad. Pol. and Hr. V. M. present in all muscles reacting. E. R.: F. absent in all of the flexors of the fingers.

Note.—This case shows the phenomenon of difference in the long flexors of the fingers.

Case 16.—A. G. (Figs. 16, 41); wd. July 18, 1918; op. May 24, 1919, C. Diag.: Partial paralysis of the left median nerve. Nerves Stimulated and Muscles Reacting: median: F. C. R.; ulnar: Hr., I., Ad. Pol., F. C. U. and F. P. D. V. M. present in all muscles reacting.

Case 36.—L. I. (Fig. 17); wd. Sept. 28, 1918; op. June 10, 1919. Diag.: Complete paralysis of the left median and ulnar nerves, lateral neuromas on both. The P. R. T. reacted through the median at op.

Note.—This case shows the phenomenon of difference in the P. R. T.

Case 44.—D. P. (Fig. 18); wd. Oct. 8, 1918; op. July 19, 1919. Diag.; Neuroma of the median and compression of the ulnar nerves. Nerve Stimulated: median. Muscles Reacting: P. R. T., F. S D. and F. C. R.

Case 13.—G. C. K. (Figs. 19, 44); wd. Oct. 8, 1918; op. May 22, 1919, F. Diag.: Paralysis of the left ulnar and median nerves (incomplete in the latter), both nerves embedded in scar tissue. Nerves Stimulated and Muscles Reacting: median, P. R. T., F. C. R., P. L. and F. P. D.; ulnar, F. P. D. V. M.: limited to pronation and flexion of the wrist. E. R.: F.; there were no reac-

tions previous to operation, in any of the ulnar muscles, and none in the median below the palmaris longus. G.: slow reaction without polar reversal in F. P. D. and F. C. U. and with reversal in the intrinsic hand muscles.

Note.—Stimulation of the ulnar exposed at op. in this case gave a reaction in F. C. U. when the nerve was stimulated below the lesion, but not from above it—a direct paradoxical phenomenon.

Case 3.—C. W. (Figs. 20, 46); wd. Oct. 4, 1918; op. May 7, 1919, C. Diag.; Partial paralysis of the ulnar nerve. Nerves Stimulated and Muscles Reacting: median, P. R. T., F. C. R. and F. L. P.; ulnar, F. C. U. and F. P. D. V. M.: included slight movement in the F. C. U. E. R.: F.; no reaction in ulnar distribution. G.: slow contractions with polar reversals in the ulnar muscles.

Note.—This case shows the phenomenon of difference between direct and percutaneous stimulation in the F. C. U. and F. P. D. The case is also interesting as showing the predominance of reactions in the proximal muscles of a normal nerve, the median.

Case 26.—T. F. (Fig. 21); wd. July 20, 1918; op. May 31, 1919, C. Diag.: Partial paralysis of the left median nerve, lateral neuroma. Nerve Stimulated and Muscles Reacting: median, P. R. T., F. C. R., F. L. P. and F. P. D.

Note.—The phenomenon of difference between direct and percutaneous stimulation was demonstrated in this case, which is also of special interest since it was possible to obtain reactions of the P. R. T. from the external side of the nerve above the lesion, while an inch or so below a similar reaction was obtained from the inner aspect of the nerve, indicating that the lesion lay just at the point of crossing of the fibers supplying the pronator.

Case 9.—M. P. H. (Fig. 22); wd. July 15, 1918; op. May 5, 1919, F. Diag.: Paralysis of the median and ulnar nerves; both were embedded in scar tissue. Nerve Stimulated: median, muscles reacting, F. C. R., F. P. D., P. R. T. and F. L. P. V. M.: limited to contractions of the P. R. T. and F. C. R. E. R.: F.; no reactions. G.: slow reactions with polar reversal in the muscles affected.

Note.—The phenomenon of difference was demonstrated in this case.

Case 67.—C. K. (Fig. 23); wd. Oct. 19, 1918; op. Aug. 21, 1919, Kg. Diag.: Paralysis of the left median nerve. The median nerve was stimulated and the muscles reacting were the P. R. T. and F. C. R.

Case 50.—W. E. T. (Figs. 24, 47); wd. Oct. 16, 1918; op. July 30, 1919, Kg. Diag.: Paralysis of the left median and ulnar nerves, both of which were embedded in scar tissue. Nerves Stimulated and Muscles Reacting: median, P. R. T., F. C. R. and F. P. D.; ulnar, F. C. U.

Case 53.—F. H. M. (Fig. 25); wd. Sept. 27, 1918; op. Aug. 4, 1919, Kp. Diag.: Paralysis of the left median nerve. This nerve was stimulated with resulting reactions in the P. R. T., F. C. R. and F. P. D. muscles.

Case 19.—F. E. C. (Figs. 26, 27, 28); wd. Nov. 1, 1918; op. May 27, 1919, F. (local anesthesia). Diag.: Paralysis of the right median and ulnar nerves; causalgia of the median. The median nerve was stimulated at operation, giving reactions in P. Q., O. P. Ab. Pol., first and second Lum. The sensory reaction was described by the patient by the words "it hurts" and was localized in all the fingers except the fifth.

Case 30.—W. B. (Fig. 29); wd. July 30, 1918; op. June 4, 1919. Diag.: Paralysis of the right median nerve below the branches to the forearm muscles. Nerve Stimulated and Muscles Reacting: median, F. C. R. and P. L. The stimulation was applied below the point of offset of the branch to the P. R. T., which was seen leaving the nerve from its inner side.

CASE 69.—S. K. (Fig. 30); wd. Sept. 2, 1918; op. Sept. 30, 1919, Kg. Diag.: Partial paralysis of the right median nerve. This nerve was stimulated at operation and reactions obtained in the P. Q. and F. P. L. muscles.

CASE 27.—J. T. W. (Figs. 31, 32, 33, 57); wd. Nov. 19, 1918; op. June 19, 1919; local anesthesia. Diag.: Partial paralysis of the median and ulnar nerves; neuromas. Only sensory reactions were obtained.

CASE 17.—D. D. (Figs. 34, 35, 36); wd. Oct. 1, 1918; op. May 26, 1919, C., under local anesthesia. Diag.: Paralysis of the median nerve at the wrist; anatomic division with proximal neuroma. Only sensory reactions were obtained, the patient complaining of "tingling" and "burning" when the proximal stump was stimulated.

CASE 60.—J. J. (Fig. 45); wd. Oct. 10, 1918; op. Aug. 8, 1919, Kg. Diag.: Partial paralysis of the right ulnar nerve; neuroma. Reaction was obtained in the F. C. U. muscle.

CASE 66.—P. F. (Fig. 48); wd. Oct. 18, 1918; op. Aug. 19, 1919, Kp. Diag.: Partial paralysis of the left ulnar nerve. Reactions were obtained in the F. C. U., F. P. D., Io., Ad. Pol. and Ab. M. D.

CASE 42.—W. G. (Fig. 49); wd. Oct. 18, 1918; op. June 25, 1919, C. Diag.: Partial paralysis of the left ulnar nerve; neuroma Muscles reacting: F. P. D. and F. C. U.

CASE 28.—E. P. (Fig. 50); wd. Nov. 3, 1918; op. June 3, 1919, F. Diag.: Compression of the left ulnar nerve below branches to the forearm muscles. Reactions were obtained in the Ad. Pol. and Io. from stimulation below but not from above the lesion on the exposed nerve.

Note.—An example of the direct paradoxical phenomenon.

CASE 8.—P. B. (Fig. 51); wd. Sept. 26, 1918; op. May 15, 1919, C. Diag.: Complete lesion of the right ulnar nerve below the branch to the F. C. U.; separation of 3 cm. with proximal and distal neuromas. Muscles reacting F. C. U. and F. P. D. E. R.: F.; reactions in the ulnar distribution limited to the F. C. U. G.; F. P. D. gave a slow reaction without polar reversal.

Note.—The reactions were obtained by stimulating the branches to the muscles, not the main trunk. These branches left the nerve from its internal aspect. The case shows the difference between direct and percutaneous stimulation in the reactions of the F. P. D.

CASE 47.—J. F. B. (Fig. 52); wd. Oct. 14, 1918; op. July 15, 1919, C. Diag.: Partial paralysis of the left ulnar nerve; only a small fasciculus on the inner border of the nerve was not severed. Reactions obtained in the Io.

CASE 49.—A. F. J. (Fig. 53); wd. June 19, 1918; op. June 29, 1919, F. Diag.: Partial paralysis of the left ulnar nerve; fusiform neuroma at the site of a neurorrhaphy performed Dec. 3, 1918. (The second operation was done because there was no improvement after the first.) V. M.: none in any of the ulnar hand muscles. E. R.: F.; no reaction in ulnar hand muscles. The intrinsic muscles of the hand supplied by the ulnar nerve reacted from stimulation of the exposed nerve at operation.

Note.—This example of the phenomenon of difference occurred eight months after complete division and suture of a nerve, and must have been due to partial regeneration which had not shown itself in a return of voluntary movement.

CASE 18.—M. C. M. (Fig. 54); wd. July 20, 1918; op. May 23, 1919, F. Diag.: Lesion of the musculospiral nerve. No reactions were obtained from

this nerve, but the ulnar nerve which was normal was exposed and stimulated. The muscles reacting were: Ad. Pol., F. C. U. and F. P. D.

CASES 34 AND 35.—(Figs. 55, 56, 57). These cases are included since in the former the branches to the F. C. U. and F. P. D., and in the latter branches to the F. C. U. were clearly seen leaving the ulnar nerve from the inner side. They were stimulated in each instance, confirming the observations.

CASE 39.—J. W. W. (Fig. 60); wd. Nov. 1, 1918; op. June 19, 1919, F. Diag.: Paralysis of the inner cord of the brachial plexus. The circumflex nerve was stimulated at operation, the deltoid muscle reacting. V. M.: slight in the deltoid. E. R.: F.; none in deltoid. G.; slow wavy without polar reversal in the deltoid.

Note.—This case showed the phenomenon of difference between direct and percutaneous stimulation.

CASE 33.—G. R. C. (Fig. 62); wd. Nov. 5, 1918; op. June 6, 1919, C. Diag. paralysis of the musculospiral nerve below branches to the triceps; neuroma. Nerve Stimulated: musculospiral. Muscle Reacting: triceps.

CASE 15.—E. D. (Fig. 63); wd. Aug. 6, 1918; op. May 23, 1919, F. Diag.: Paralysis of the musculospiral nerve below branch to triceps; neuroma. Nerve Stimulated: musculospiral. Muscle Reacting: extensor indicis. V. M.: none in musculospiral distribution. E. R.: F.; none. G.; slow reaction without polar reversal.

Note.—Similar responses were obtained at operation by stimulating the nerve above and below the lesion, both before and after section of the nerve. The case is an example of the phenomenon of difference between direct and percutaneous stimulation.

CASE 22.—A. J. B. (Fig. 64); wd. Oct. 6, 1918; op. May 29, 1919, F. Diag.: Compression of the right musculospiral nerve below the branch to the E. C. R. Nerve Stimulated: musculospiral. Muscle Reacting: E. C. R.

CASE 20.—R. M. S. (Fig. 66); wd. Oct. 14, 1918; op. May 27, 1919, C. Diag.: Paralysis of the left external popliteal nerve; neuroma. Nerve Stimulated: Ext. Pop. Muscles Reacting: H. S., Gast., F. L. H., F. L. D. and T. P.

CASE 23.—F. S. (Fig. 67); wd. Sept. 29, 1918; op. April 4, 1919, C. Diag.: Paralysis of the left sciatic nerve; neuroma. Nerve Stimulated: sciatic. Muscle Reacting: H. S.

CASE 24.—W. H. H. (Fig. 68); wd. July 18, 1918; op. April 22, 1919, F. Diag.: Paralysis of the right sciatic nerve, partial; lateral neuroma on outer side. Nerve Stimulated: sciatic. Muscles Reacting: Gast. and Sol. group. V. M., slight flexion of great and fifth toes; E. R. F., none below knee; G., wavy contraction without reversal in internal popliteals, and with reversal in external popliteal muscles.

Note.—We are indebted to Lieutenant-Colonel Frazier for the notes on this case. It is an example of the phenomenon of difference between direct and percutaneous stimulation as the gastrocneumeus and soleus reacted from direct stimulation of the nerve at operation, but had failed to react from stimulation through skin.

CASE 64.—B. (Fig. 69); wd. Sept. 27, 1918; op. Aug. 16, 1919, Kg. Diag.: Partial paralysis of the external division of the left sciatic nerve; neuroma. Nerve Stimulated: sciatic (Int. Pop. division). Muscles Reacting: Bi., Gast., Sol and T. P.

CASE 12.—A. G. (Fig. 70); wd. Aug. 9, 1918; op. May 21, 1919, C. Diag.: Partial paralysis of Int. and Ext. Pop. nerves. Nerve Stimulated: Int. Pop. Muscle Reacting: Gast. and Sol. group, T. P., F. L. D. and F. L. H.

CASE 14.—S. H. (Figs. 71 and 90); wd. Sept. 29, 1918; op. May. 22, 1919, C. Diag.: Paralysis of the right sciatic nerve; anatomic division of the Int. Pop. portion of the nerve. Nerve Stimulated: Ext. Pop. portion of the sciatic. Muscles Reacting: T. A., Per. and extensors of the toes. V. M.: slight movement of the muscles supplied by the Ext. Pop. nerve. E. R. F.; weak reactions in same muscles. G.: slow reaction without polar reversal.

Note.—This case is interesting in showing the difference in position of the fasciculi which supply the peronei in the trunk of the sciatic and in that of the Ext. Pop. nerve. They lie on the posterior-internal aspect of the nerve, which only permits stimulation from the posterior aspect of the sciatic, but when the external division is separated these fasciculi can be stimulated both posteriorly and internally.

CASE 21.—L. E. (Fig. 72); wd. Sept. 29, 1918; op. May 28, 1919; C. Diag. Partial paralysis of the left sciatic nerve; neuroma on int. div. Nerve Stimulated: Ext. Pop. div. of sciatic. Muscles Reacting: peronei. V. M.: none. E. R.: F.: none. G.: slow reaction without polar reversal.

Note.—This is another example of the phenomenon of difference between direct and percutaneous stimulation.

CASE 59.—J. J. (Figs. 73, 93); wd. July 20, 1918; op. Aug. 8, 1919, Kg. Diag.: Incomplete paralysis of the left sciatic nerve; causalgia; compression by scar tissue. Nerve Stimulated: sciatic (ext. portion). Muscles Reacting: Per. and T. A.

Note.—This case is of interest in showing the apparent rotation of the peronei fasciculi. The results of stimulation of the external popliteal alone differ somewhat from those in Case 14. However, the former stimulation was applied at a point above the bifurcation after the two divisions of the sciatic had been dissected apart, while in this case the stimulation was applied near the head of the fibula. The nerve makes a twist at about this place which probably accounts for the seeming inconsistency.

CASE 1.—F. M. (Fig. 75); wd. Sept. 30, 1918; op. April 29, 1919, F. Diag.: Partial paralysis of the right sciatic nerve; neuroma of the external portion. Nerve Stimulated: Int. Pop. division of the sciatic. Muscles Reacting: Gast. and Sol. group and T. P.

CASE 40.—A. L. R. (Fig. 76); wd. Aug. 4, 1918; op. June 19, 1919, A. Diag.: Paralysis of the left external popliteal nerve; neuroma. Nerve Stimulated: internal popliteal. Muscles Reacting: Gast. and Sol., T. P., F. L. D. and F. L. H.

CASE 56.—G. G. (Fig. 77); wd. Sept. 29, 1918; op. Aug. 6, 1919, C. Diag.: Incomplete paralysis of the left sciatic nerve; external division embedded in scar. Nerve Stimulated: internal popliteal. Muscles Reacting: Gast. and Sol., T. P.

Note.—The internal division was separated before stimuli were applied.

CASE 63.—M. F. (Fig. 78); wd. Oct. 2, 1918; op. Aug. 14, 1919, Kg. Diag.: Incomplete paralysis of the right sciatic nerve; anatomic interruption of the external portion. Nerve Stimulated: internal popliteal. Muscles Reacting: Gast. and Sol., Bi. and T. P.

Note.—The internal division was separated before stimuli were applied.

Case 6.—F. S. (Fig. 79); wd. Sept. 27, 1918; op. May. 4, 1919, C. Diag.: Partial paralysis of the right sciatic nerve; complete division of the external popliteal and neuroma of the internal popliteal, external aspect. Nerve Stimulated: internal popliteal. Muscles Reacting: T. P., F. L. D. and F. L. H.

Case 10.—D. H. (Figs. 80, 81, 89); wd. Sept. 30, 1918; op. May 17, 1919, F. Diag.: Partial paralysis of the right sciatic nerve; neuroma of the external division. Nerve Stimulated: internal popliteal, external popliteal. Muscles Reacting: Per., Gast. and Sol., T. P., F. L. D. and F. L. H.

Note.—This case is interesting in that the external popliteal nerve was split and stimulation was applied to the cut surface. The results showed that the fasciculi to the peronei lay posteriorly.

Case 52.—N. G. (Fig. 82); wd. July 24, 1918; op. Aug. 4, 1919, C. Diag.: Partial paralysis of the right sciatic nerve; neuroma of the external popliteal and external side of the internal popliteal nerves. Nerve Stimulated: internal popliteal. Muscles Reacting: Gast. and Sol.

Case 58.—R. M. L. (Fig. 83); wd. Oct. 23, 1918; op. Aug. 7, 1919, Kg. Diag.: Paralysis of the left sciatic nerve; interruption of external division. Nerve Stimulated: internal popliteal. Muscles Reacting: Gast. and Sol., and T. P.

Case 68.—J. G. (Fig. 84); Diag.: Partial paralysis of the sciatic nerve. V. M.: slight in muscles of the distribution of the internal popliteal nerve. Nerve Stimulated: internal division of the sciatic. Muscles Reacting: Gast. and Sol., T. P. and intrinsic muscles of the foot.

Case 7.—O. C. S. (Fig. 85); wd. Aug. 4, 1918; op. May 14, 1919, F. Diag.: Paralysis of the left external popliteal nerve; anatomic division. Nerve Stimulated: internal popliteal. Muscles Reacting: Gast. and Sol., T. P., F. L. D. and F. L. H.

Case 51.—S. S. (Fig. 86); wd. Oct. 10, 1918; op. July 30, 1919. Diag.: Paralysis of the internal popliteal nerve; compression by scar tissue. Nerve Stimulated: internal popliteal. Muscles Reacting: intrinsic muscles of the foot.

Case 54.—A. S. T. (Fig. 87); wd. Sept. 28, 1918; op. Aug. 5, 1919, Kp. Diag.: Partial paralysis of the internal popliteal nerve; neuroma; causalgia. Nerve Stimulated: internal popliteal Muscles Reacting: T. P. and F. L. D.

Case 46.—F. O. (Fig. 91); wd. Aug. 3, 1918; op. July 24, 1919. Diag.: Partial paralysis of the right external popliteal nerve; neuroma involving the nerve with the exception of a few fasciculi on the posterior-external aspect. Nerve Stimulated: external popliteal. Muscles Reacting: the peronei.

Note.—The results in this case do not correspond to the others for this group of muscles, probably because the nerve was so extensively involved and may have been rotated by the injury.

Case 38.—J. R. (Fig. 92); wd. Oct. 9, 1918; op. June 17, 1919. Diag.: Paralysis of the left anterior tibial nerve; lateral neuroma. Nerve Stimulated: anterior tibial. Muscles Reacting: peronei.[10]

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10. In addition to the references already given, the following may be of interest:

Sherren: Injuries of Nerves and Their Treatment, New York, William Wood & Co., p. 196.

Platt and Brentnall: Faradic Stimulation of Nerve and Muscle During Operations, Lancet **1**:884 (May 24) 1919.

SOME OBSERVATIONS ON EPILEPTICS AND ON EPILEPSY, CHIEFLY FROM A ROENTGEN-RAY STANDPOINT

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At the fortieth annual meeting of the American Neurological Association, held in May, 1914, a paper was presented, entitled "Observations on Epilepsy Chiefly from an X-Ray Standpoint," by Drs. T. M. T. McKennan, George C. Johnston and C. H. Henninger. In that paper it was shown that there were apparently deposits of bone in the base of the skull in a large percentage of epileptics. An endeavor was made to show the way in which this deposit occurred and that, together with this deposit, there was probably a crippling of the pituitary gland. To make this clear I quote:

> The interpretation of the changes found at the base of the skull in the essential epilepsies at first seemed to us to be very difficult. Our first idea was that it had an inflammatory origin. This we were forced to abandon for the reason that we could find no cause of an infective character that would produce inflammation, for the relative regularity of the changes in certain situations in the cases examined. The viewpoint that the bony overgrowth is a local acromegaly and that the bony deposit is brought about through venous congestion at the base of the skull seems to us to be the only logical explanation.
>
> To comprehend this a knowledge of the venous circulation at the base of the skull is essential. The veins from the anterior lobe of the pituitary gland empty into the intercavernous sinuses and the secretion from the anterior lobe of the pituitary is poured into these sinuses. The intercavernous sinuses vary in number from one to three; there is probably an average of two. Knott found a single intercavernous sinus in twelve out of forty-four examined. In fifteen out of forty-four he found two. Most textbooks of anatomy give these two intercavernous sinuses the name of the circular sinus. It should, however, be noted that the intercavernous sinuses are really veins and not sinuses. Hence they are collapsible.
>
> These intercavernous veins serve to join the two cavernous sinuses and they receive also a number of small veins from the adjacent parietal dura mater. This dura, as we know, is the internal periostium. These dural veins drain an area of the base of the skull comprising the clinoid processes and the dorsum sella, the anterior fossa and the body of the sphenoid. The anastomosis of these veins with other veins probably varies considerably. The bony deposit in epileptics corresponds to the areas drained by these dural veins of the base and leads directly to the conviction that the process of bony change can only be brought about by some pressure within the small confined boxlike space that contains the pituitary gland as well as the intercavernous veins.
>
> Pressure within this boxlike space that would prevent the intercavernous veins from discharging their contents into the cavernous sinuses would cause

the blood in this boxlike structure to be backed into the dural veins. The circulation at this point would be reversed and the blood loaded with secretion from the anterior lobe of the pituitary gland would bathe the bony structure of the base of the skull in the region of the clinoid processes, dorsum sella, the anterior fossa and the body of the sphenoid bone. Increased bony deposit would consequently be the result. This deposit, however, would vary in amount depending on two factors; first, the duration of pressure that causes the reversal of circulation, and second, on the freedom of anastomosis of the dural veins.

The next question that logically arises is: What can cause the pressure within this boxlike structure? Hypertrophy of the pituitary gland is improbable for this condition has not been found in postmortem examinations of epileptics. It seems more probable to us that it is intermittent hyperemia, and this viewpoint is somewhat confirmed by the fact that postmortem examinations of epileptics reveal abnormally large veins within the pituitary gland.

The question then naturally arises: What is the cause, or what can be the cause of intermittent hyperemia in this situation? Does it occur from the attack of epilepsy with the attendant general venous congestion? We think this extremely improbable, for our cases of symptomatic epilepsy showed no bony deposit. Again, hyperemia, the result of an attack of epilepsy, would naturally last for a few minutes only and the blood stream would soon return to its normal course. Hyperemia would have to exist for hours or days to permit of any bony deposit.

It seems more probable that the venous hyperemia would be due to some vasomotor disturbance of the anterior lobe of the pituitary gland. This same venous hyperemia would doubtless tend to prevent a proper amount of secretion of the posterior lobe from gaining access to the third ventricle by way of the infundibulum and in all probability repeated hyperemias would cause a degree of crippling of the entire pituitary gland, the posterior lobe, as well as the anterior lobe.

CASES STUDIED BY AUTHOR

The present study comprises ninety cases.

TABLE 1.—RESULTS OF THE ROENTGEN-RAY FINDINGS

	No. of Cases	Per Cent.
Tumors or evidence of pressure in the interpituitary area	9	10.
Bony deposits	52	57.77
Small areas	10	11.
Calcareous degeneration	2	2.5
Cerebropathy (this is not shown by the roentgen ray)	9	10.
No changes	8	8.8
Total	90	100.

The designation "small area" may be explained thus: In some cases the pituitary area appears to be very small. It has the appearance of being infantile, and we designate it a small or infantile area. It would be a strong argument in favor of a small pituitary gland within this area—a gland that possibly has not increased in size commensurately with bodily growth and, therefore, probably an inefficient gland. We cannot accurately measure this area and say definitely

what size the area should be to be normal, but by experience with roentgen-ray plates we can judge what appears to be normal and what appears to be abnormal. In many cases with increased bony deposit I have noted a pituitary area that appears to be small, and it is my belief that an increasing number will be found to have abnormally small pituitary areas. As to the extent of the bony deposits or bony overgrowth, I am now quite satisfied that it may occur in almost any part of the skull and that the situation of the main deposit is governed entirely by the extent and kind of the venous anastomosis that exists in the individual case. I am coming to the belief that essential epilepsy is largely due to a crippling of the pituitary gland in one way or another. I believe that the most frequent cause is probably recurring hyperemia of the gland; that second in importance is a gland that has not evoluted properly—a small inefficient gland; and third in importance is pituitary tumor or struma, or in some cases probably simple hypertrophy. The question of abnormality of the pituitary gland in its relation to epilepsy is greatly illuminated by a consideration of cases of epilepsy originating in persons over 35 years of age. (These cases were taken from my total number of cases in which I have roentgen-ray plates).

TABLE 2.—Cases Starting in Persons Over 35 Years of Age

	No. of Cases	Per Cent.
Tumor	13	48.1
Bony deposits	7	25.9
Small area	4	14.8
Cerebropathies	3	11.2
Total	27	100.

It is well known that tumor of the pituitary gland usually disturbs the function of that gland. Epilepsies in these cases are regarded as due to disturbance or destruction of the function of this gland. If we accept the theory that the small area is an evidence of pituitary inefficiency, and further, if we accept the theory that increased bony deposits are an evidence of pituitary crippling, it would be shown by the figures above that in cases of epilepsy originating in persons over 35 years of age, 88 per cent. present roentgen-ray evidence of pituitary inefficiency.

To view the matter from another angle, let us leave out of consideration the cases of cerebropathy for the time. Thus there are twenty-four cases of epilepsy starting in persons over 35 years of age. More than 50 per cent. of these cases being known to be due to an organic affection of the gland, causing it to be inefficient, would be

a strong argument in itself for the conclusion that the other cases must have inefficient pituitary glands from some cause or causes.

TABLE 3.—Epilepsy Beginning in Persons Over 35 Years of Age

	No. of Cases	Per Cent.
Tumor	13	53.63
Bony deposits	7	29.87
Small area	4	16.50
Total	24	100.

In some cases in which there was evidence of pressure in the pituitary area I have been able to have a roentgenogram made again in one or more years and have, in some instances, found that there was no progression. It would seem that if these cases are struma or other tumor growths the process of enlargement had ceased. It is possible that some of them may be simple hypertrophy.

The question of pituitary feeding in epileptics, in my opinion, is an important one. I have found that the extract of the whole gland in 2 grain doses, three times a day, and given four hours after eating, is the most satisfactory. Bromids should always be used in conjunction with the pituitary extract. I am in the habit of using from 45 to 60 grains of bromid daily. After one year less bromid can be used, but the pituitary feeding must be kept up indefinitely, probably during the life of the patient.

CONCERNING THE COLLOIDAL MASTIC TEST*

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The reported results of the application of the colloidal mastic test to the examination of pathologic spinal fluids have been so contradictory that it has appeared wise to conduct an investigation to determine the degree of parallelism, in regard to their clinical value, between the colloidal gold and the colloidal mastic reactions, and the possible cause for a discrepancy, if any exists.

HISTORICAL NOTE

The application of colloidal solutions to the examination of the spinal fluid dates from the work of Lange,[1] who made use of a colloidal gold solution. Neisser and Friedemann,[2] among others, demonstrated the similarity in action between soluble proteins and a colloidal gold or mastic solution. Emmanuel,[3] in 1915, published his notes on the application of the colloidal mastic test to the examination of various pathologic spinal fluids. In brief, the technic used by Emmanuel in employing a colloidal mastic solution is as follows: One and five tenths c.c. of a 1.25 per cent. sodium chlorid solution is placed in the first of five tubes and 1 c.c. is placed in each of the remaining tubes. Five tenths c.c. of the spinal fluid to be tested is placed in the first tube, the contents are well mixed and 1 c.c. is transferred to the second tube and so on. The 1 c.c. of fluid removed from the fourth tube is discarded, the fifth tube serving as a control. One c.c. of the mastic emulsion (prepared as will be described) is now added to each tube, the tubes are shaken and allowed to stand for twelve hours. Abnormal fluids give a precipitation in varying degree in some or all of the tubes.

Of the thirty-two fluids examined by Emmanuel, precipitation occurred in all the dilutions prepared from the fluids of paretics, tabetics and cerebrospinal syphilitics. It was found that frequently

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1. Lange, C.: Die Ausflockung Kolloidalen Goldes durch Cerebrospinalflüssigkeit bei leutischen Affektionem des zentralnervensystem, Ztschr. f. Chemotherap. **1**:44, 1912.

2. Neisser, M., and Friedemann, O.: Studien über Ausflockungserscheinungen II, Beziehung zur Bakterienagglutination, München. med. Wchnschr. **51**:827, 1904.

3. Emmanuel, G.: Eine neue Reaktion zur Untersuchungen des Liquor Cerebrospinalis, Berl. klin. Wchnschr. **52**:792, 1915.

there was some precipitation in the fourth tube, even with normal fluids. Jacobstahl and Kafka,[4] in 1918, described a slightly different technic, using two curves—depending on the salt sensitiveness of the mastic sol. For recording their results they made use of eight degrees of change in the mastic sol.

In regard to the diagnostic value of the test, one finds rather conflicting opinions. Victors,[5] using the original method of Emmanuel, reported that the technic of the test was relatively simple and that the information obtained from it was trustworthy. Lowrey[6] examined thirty-six fluids, using the original technic and decided "that it cannot obviously do the work of the gold test." Urechia and Jorgulesci[7] using the original technic decided that the mastic and the gold sol reactions gave the same results but that the mastic was not as sensitive as the gold.

In 1917, Cutting[8] reported that by the addition of a trace of potassium carbonate to the sodium chlorid solution, used for making the dilutions, the control tube showed no precipitation and the dilutions of the spinal fluid could be run out as far as desired. In the same year, Smith[9] reported the use of the Cutting modification of the test. He carried the dilution through ten tubes, but in particular, made use of a numerical nomenclature for expressing the degree of precipitation of the mastic: 1, denoted a milky fluid, loss of opalescence and a very slight precipitate; 2, a distinct precipitate but with a milky supernatant fluid; 3, a marked precipitate with a slight cloudiness of the fluid above; and 4, complete precipitation of all the colloid. Smith reported duplicate curves on the fluids of twenty-three different patients. His results show an exact agreement (as regards type of reaction) of the two curves in twenty-two of the twenty-three fluids examined. The gold curve of six of his fluids was paretic or near-paretic in type; in eleven fluids there was a change of the nonparetic variety; while six of the fluids yielded a negative result. Smith is of the opinion that the information obtained from the mastic test is the

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4. Jacobstahl and Kafka: Mastix-Reaktion, Berl. klin. Wchnschr. **55**:249, 1918.

5. Victors, E. A.: Colloidal Mastic Reaction of Cerebrospinal Fluid, California State J. M. **16**:32, 1918.

6. Lowrey, L. G.: Mastiche and Potassium Permanganate Tests Applied to the Cerebrospinal Fluids of the Insane, Boston M. & S. J. **177**:557, 1917.

7. Urechia, C. J., and Jorgulescu, N.: L'epreuve colloidale au mastic d'Emmanuel dans le liquide cephalo-rachidien, Compt. rend. Soc. de biol. **79**: 893, 1916.

8. Cutting, J. A.: A New Mastic Test for the Spinal Fluid, J. A. M. A. **68**:1810 (June 16) 1917.

9. Smith, E. R.: The Mastic Reaction on the Cerebrospinal Fluid, Med. Rec. **92**:675, 1917.

same as that obtained from the gold test. Immermann [10] in his report on the mastic test concluded that "the gum mastic test is not equivalent to nor supplementary to the colloidal gold reaction." His clinical material consisted of the fluids from twenty cases. If Smith's numerical nomenclature be applied to Immermann's description of his curves, and the gold and mastic curves be graphically plotted and compared, it will be found that with three of the fluids the curves were relatively identical, with five of the fluids they were similar in some respects, while with the remaining twelve fluids they were dissimilar.

Smith and Lowrey,[11] after the experience resulting from the examination of 268 fluids by the Cutting modification of the Emmanuel test, felt that "the mastiche reagent is simple and the interpretation is easy. Furthermore, it seems to be very accurate." These workers, however, did not make use of a numerical system for recording their results but simply reported the mastic test as positive or negative. With no case of the 268 was the mastic test positive when the gold was negative.

Langdon,[12] after the examination of a series of fluids by both colloidal reactions, was of the opinion that the mastic test was "probably not as delicate or valuable" as the gold test. He stated that the gold test indicated whether the curve was of the paretic, syphilitic or meningitic type while the mastic test showed merely a positive or negative result. Langdon made use of no numerical method of recording the changes in the various tubes of the mastic test.

METHODS USED

Preparation of Mastic Emulsion.—Ten grams of gum mastic were dissolved in 100 c.c. of absolute alcohol. The resultant cloudy solution was filtered several times through paper, until a brilliant straw colored fluid was obtained. This solution was kept in glass stoppered bottles in the open laboratory, but never in the direct sunlight. The emulsion was prepared from this stock solution thus: To 1 c.c. of the stock solution in a test tube, 9 c.c. of absolute alcohol were added. Forty c.c. of distilled water were placed in a small Erlenmeyer flask and the mastic solution in alcohol was then rapidly poured in and the whole gently mixed by rotation.

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10. Immerman, S. L.: The Mechanism and the Significance of the Gum Mastic Test, J. A. M. A. **69**:2027 (Dec. 15) 1917.

11. Smith, C. E., and Lowrey, L. G.: On the Use of the Emmanuel-Cutting Mastiche Test in Examining Spinal Fluids from Psychopathic Subjects, Boston M. & S. J. **177**:557, 1917.

12. Langdon, F.: The Mastic Test for the Diagnosis of Cerebrospinal Syphilis, J. Lab. & Clin. M. **3**:376, 1918.

Preparation of the Dilutions.—Ten small test tubes were set up in a rack. To the first were added 1.5 c.c. of the salt solution (99 c.c. of 1.25 per cent. sodium chlorid solution plus 1 c.c. of 0.5 per cent. potassium carbonate solution), and to each of the remainder 1 c.c. was added. Five tenths c.c. of the spinal fluid was added to the first tube, and after mixing, 1 c.c. was transferred to the second tube; this was mixed, and so on. To each tube 1 c.c. of the mastic emulsion was added; each tube was shaken just enough to secure even distribution; the tubes were allowed to stand over night and were then read. The results were expressed according to Smith nomenclature. The gold tests were carried out according to the usual standards.

COMPARISON OF THE GOLD AND MASTIC CURVES OF THE FLUIDS EXAMINED

The comparative curves presented in this paper are 100 in number and form a consecutive series of the fluids examined. These 100 fluids exhibited a paretic gold curve in sixteen instances, a positive

TABLE 1.—THE MASTIC REACTION ON FLUIDS HAVING A PARETIC TYPE OF COLLOIDAL GOLD REACTION

Fluid No.	Diagnosis	Gold	Mastic	Cell Count	Pandy Test	Cerebrospinal Fluid Wassermann Reaction
1	Paresis	5554344300	4444431000	34	+	++++
2	Paresis	5555320000	3444200000	60	..	++++
3	Paresis	5542000000	4431000000	36	..	++++
4	Syphilitic myelitis	5543100000	4431000000	14	+	++++
5	Paresis	4543100000	4432100000	12	+	++++
6	Paresis	5555420000	4444310000	23	+	++++
7	Paresis	5542100000	4432000000	6	+	++++
8	Hemiplegia	5554100000	2322000000	74	+	++++
9	Brain tumor	5555433210	4444443210	6	+	—
10	Lethargic encephalitis	5554320000	4444431000	6	+	—
11	Paresis	5555554310	4444310000	..	..	++++
12	Paresis	5555542100	4443210000	..	+	++++
13	CNS syphilis	5443210000	4431000000	20	+	++
14	Paresis	5210000000	4432000000	..	+	++++
15	Paresis	5554321000	3444321000	153	+	++++
16	Paresis	4432100000	3432000000	140	+	++++

gold curve but not of the paretic variety in fifty-five instances, and in twenty-nine instances gave a negative reaction. For comparative study the fluid findings have been tabulated in these three groups on the basis of the gold test.

Bearing in mind that the maximum change in the gold test is represented in the nomenclature by 5 and that the maximum change in the mastic test is represented by 4 and that lesser numerical values differ proportionately, it is evident from a study of Table 1 that the mastic curve on eleven of the sixteen fluids embodied therein pointed to the same clinical condition as did the gold curve. On fluids number 1 and 5, the gold curves were slightly atypically paretic while the

mastic curves were typically paretic and the patients were undoubted paretics. On fluid number 2 the mastic curve was slightly atypical while the gold curve was quite typical of paresis. The gold curve of fluid number 8 was characteristic of paresis; the mastic curve,

TABLE 2.—THE MASTIC REACTION ON FLUIDS HAVING A POSITIVE BUT NONPARETIC COLLOIDAL GOLD REACTION

Fluid No.	Diagnosis	Gold	Mastic	Cell Count	Pandy Test	Cerebrospinal Fluid Wassermann Reaction
17	.....	0011132100	0024441000	..	+	
18	CNS syphilis.....	4333210000	3320000000	84	+	++++
19	Optic atrophy.....	0112100000	1210000000	46	+	−
20	Tabes.....	0111000000	2110000000	5	±	++++
21	.....	0001000000	0010000000	..	−	−
22	CNS syphilis.....	0132100000	2110000000	9	±	++++
23	CNS syphilis.....	0012100000	0121100000	..	+	
24	Optic atrophy.....	0112000000	0110000000	38	+	++++
25	Tuberculous meningitis.....	0012100000	0121100000	63	+	−
26	CNS syphilis.....	0002331000	0231000000	18	+	−
27	Poliomyelitis.....	1231000000	1210000000	..	+	−
28	Poliomyelitis.....	0120000000	2100000000	..	+	−
29	Brain tumor.....	0012100000	0121000000	3	±	−
30	Tabes.....	0123410000	2310000000	34	+	++++
31	.....	0012210000	0210000000	..	+	−
32	Tabes.....	0011000000	0011100000	..	+	
33	Tabes.....	0011000000	0111000000	..	+	
34	Tabes.....	2110000000	1332000000	3	±	++
35	Tabes.....	1112332000	0132100000	..	+	++++
36	.....	0133210000	1431000000	..	−	
37	.....	0001320000	0121000000	..	+	
38	.....	0122310000	0233100000	..	+	++++
39	.....	0001000000	0001000000	88?	+	−
40	CNS syphilis.....	0121000000	0221000000	128	+	++++
41	CNS syphilis.....	0121000000	0110000000	..	±	++++
42	Tabes.....	0001000000	0000000000	1	±	−
43	Tabes.....	0012110000	2311000000	10	+	++++
44	CNS syphilis.....	0123100000	0111100000	15	+	++++
45	Tabes.....	0013321000	1121000000	11	+	++++
46	CNS syphilis.....	1100000000	1100000000	..	..	−
47	.....	0011000000	0000000000	10	−	−
48	CNS syphilis.....	1233100000	1210000000	33	+	++++
49	Tabes.....	0122100000	0110000000	10	+	−
50	.....	0121000000	0000000000	6	−	−
51	Facial palsy.....	0122100000	1210000000	169	+	−
52	Lethargic encephalitis.....	0122000000	1110000000	0	+	−
53	Tabes.....	1243100000	1111000000	20	±	−
54	Optic atrophy.....	0123100000	1100000000	8	±	−
55	Cord tumor.....	1122222232	1134443221	9	+	−
56	Tabes.....	4332100000	4310000000	46	+	++++
57	Posterolateral sclerosis.....	0111000000	0111000000	..	−	−
58	Uremia.....	0012210000	4442000000	6	−	−
59	Tabes.....	2223310000	2221000000	15	+	++++
60	Transverse myelitis.....	0234110000	1100000000	4	+	−
61	CNS syphilis ?.....	0123300000	1320000000	3	+	−
62	Cord tumor.....	1122332100	0012100000	1	+	−
63	Tubercle of cerebellum.....	0100000000	1100000000	4	+	−
64	CNS syphilis.....	1224322100	1432000000	28	+	++
65	Paresis.....	2223433200	4443211000	21	+	++++
66	.....	0011100000	0000000000	12	±	−
67	.....	0012100000	0000000000	3	−	−
68	Transverse myelitis.....	0012200000	1100000000	5	±	−
69	CNS syphilis.....	1223321000	2310000000	45	+	++++
70	.....	1122332200	0111112100	65	+	−
71	Optic atrophy.....	3334421000	3310000000	12	+	−

however, was not the typical paretic one. The patient was hemiplegic and presented no definite mental symptoms; possibly he represented one of the so-called preparetic paretics. Fluids number 15 and number 16 were from the same patient, but were taken at different times;

the patient presented the typical mental changes seen in paresis. It is to be noted that both mastic curves and one of the gold curves were atypically paretic.

From a study of Table 1 it is seen that there is but one mastic curve, that of fluid number 8, which represented a definite deviation in type from its duplicate gold curve, and that in no instance was there a paretic mastic curve with a negative gold curve or vice versa.

Six of the fifty-five fluids reported in Table 2 gave a negative mastic test with a positive gold test. With three of these fluids the change did not amount to at most more than a borderline pathologic change, and with none of the fluids did it reach higher than a number 2 change. Three of the fluids gave a paretic type of mastic curve and a nonparetic, although positive, gold reaction. In fluid number 56 the gold curve was of a borderline paretic type, but although the patient was definitely syphilitic, there were no mental changes. Fluid number 58 presented a difference in type of reaction for which no satisfactory explanation could be found. In fluid number 65 the mastic test, although differing from the gold, represented the actual condition of the patient, who was a well advanced paretic.

TABLE 3.—The Mastic Reaction on Fluids Having a Negative Colloidal Gold Reaction

Fluid No.	Diagnosis	Gold	Mastic	Cell Count	Pandy Test	Cerebrospinal Fluid Wassermann Reaction
72 to 96	Psychoneuroses, psychoses, epilepsy, etc.	0000000000	0000000000	8 or less	—	—
97	Syphilis	0000000000	0100000000	7	—	—
98	Syphilis	0000000000	0000010000	3	—	—
99	"Headache"	0000000000	0100000000	3	—	—
100	Tabes	0000000000	1000000000	6	—	—

Table 3 shows that twenty-seven fluids were normal as regards cell count, Pandy test, and Wassermann reaction and gave negative curves with both gold and mastic tests. With four of the fluids giving a negative gold test there was a slight change in one of the tubes of the mastic test. Two of the fluids forming this group were from patients having a positive blood Wassermann test. Another of the fluids was from a patient presenting the clinical picture of tabes and suffering with attacks of abdominal pain. It is obvious, therefore, that of the 100 fluids examined the mastic test was never definitely positive when the gold test was negative. It was negative with five fluids when the gold test was mildly or doubtfully positive (i. e., tests with a maximum change of a number 2 or less). It is to be noted that from the standpoint of a diagnostic or etiologic aid, the mastic reaction is nearly, if not equally, as valuable as the gold test. I feel that

those types of change other than that called the paretic type do not permit one to say definitely that a fluid is a syphilitic one, a meningitic one, etc. The so-called paretic curve is diagnostic of paresis only when accompanied by a positive fluid Wassermann reaction. The results of colloidal examinations of the spinal fluid should be considered from a relatively broad aspect and not as a means of pointing conclusively to an anatomic or etiologic diagnosis, or as a criterion to be used for determining the therapeutic value of a method of treatment.

LIMITING FACTORS OF THE MASTIC TEST

The limiting factors have been found to be primarily: the length of time that can elapse between the time the fluid is drawn and the time it will first give a curve differing from that made with the fresh fluid; the length of time that the mastic emulsion can be preserved for use; the effect of admixture of blood; and finally, a point to be considered later in explanation of the discrepant results of certain investigators of this test.

In general it may be said that the results obtained from fluids kept in a sterile manner, at room temperature, for longer than forty-eight hours, are of doubtful value. In certain instances fluids have been found—usually paretic fluids—that will give, after standing a week, a curve exactly similar to that made when fresh.

The use of old mastic emulsions did not appear to be as deleterious as that of old specimens of fluid. The mastic emulsion after standing for some time, gradually lost its opalescency, and it appeared to be wise to use an emulsion made up within the week.

Fluids containing blood, in macroscopic quantities, were found to be as entirely unsuitable for the mastic test as for the gold test.

EXPLANATION OF DISCREPANT RESULTS OF SOME OF THE PREVIOUS INVESTIGATORS

At the beginning of this work various specimens of mastic were examined regarding their suitability for the test. Six lots of mastic were examined, four from local firms and two from out of state firms. Two of these lots of mastic were found to be entirely unsatisfactory for use, giving both quantitatively and qualitatively many false positive curves, as compared with the gold curves of the same fluids. All of the samples of mastic had about the same general appearance. It is evidently only by trial that one may be certain of a suitable supply of mastic. This unsuitability of certain samples of mastic is suggested as a possible source of the discrepant results reported by certain investigators.

CONCLUSIONS

1. The results of the investigators who have made use of the mastic test (Emmanuel-Cutting) have been relatively discordant.

2. The examination of fluids by both the colloidal gold and mastic tests has indicated that the information gained from the mastic test is in close agreement with that obtained from the gold test.

3. Certain samples of gum mastic have been found to be entirely unsuited for use in performing the mastic test. This may serve to explain the conflicting reports of certain workers with this test.[13]

13. The recent article by Camp on "Colloidal Mastic Test on Cerebrospinal Fluid" (Am. J. Syphilis 4:301, 1920), which is in substantial agreement with the conclusions reached herein, came to my attention after this article had been submitted for publication.

THE PHYSIOLOGIC SIGNIFICANCE OF THE BABINSKI TOE RESPONSE *

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The abnormal plantar response that occurs in disease of the pyramidal tract, as described by Babinski,[1] consists of a group of simultaneously occurring movements, the most conspicuous of which are dorsiflexion of the big toe, separation—generally associated with plantar flexion of the other toes and eversion of the foot. The reflexes with which the phenomenon should properly be identified are, therefore, the coordinate or purposive reflexes. These reflexes, in addition to exhibiting themselves in simultaneous contraction of a large number of muscles, are like the Babinski response in that they occur exclusively or are especially prominent after the cerebral influence has been removed. Sherrington [2] and Riddoch [3] give details concerning these reflexes in the "'spinal" animal and "spinal" man.

PREVIOUS INTERPRETATIONS OF THE BABINSKI TOE RESPONSE

Among the explanations of the origin and mechanism of the toe response are: Knapp [4] attributed the dorsiflexion of the big toe to a greater permeability of the neuron paths of the extensor longus hallucis. Van Woerkom [5] considered this movement of the big toe a manifestation of a primitive function existing when man, in his evolution, abandoned an arboreal life to lead one of terrestrial progression. Marie and Foix,[6] noting that with extension of the big toe there is a shortening of the stimulated limb, concluded that the reflex represents the flexion phase of the step, or of the "mark-time" movements of Freusberg, Sherrington and others. They thus consider the Babinski toe response to be the "minimal motor response" of that phase of the step in which the limb is advancing to a new position.

Walshe,[7] who studied the flexion reflex, flexor spasms and the Babinski toe reflex, maintains that all these phenomena are identical

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1. Babinski, M. J.: Compt. rend. Soc. de biol., 1897; Semaine méd. **17**: 321, 1898.
2. Sherrington, C. S.: J. Physiol. **5**:28, 1910.
3. Riddoch, George: Brain **40**:264, 1917.
4. Knapp, P. C.: Rev. Neurol. & Psychiat. **5**:825, 1907.
5. Van Woerkom: Rev. Neurol. **22**:285, 1912.
6. Marie and Foix: Rev. Neurol. **23**:657, 1912.
7. Walshe, F. M. R.: Brain **37**:269, 1915.

in character. The toe response is thus part of that type of flexion reflex which is purely defensive, such as the withdrawal of the limb on the application of a hurtful stimulus. Whereas Marie and Foix look upon the toe response as the "minimal motor response" in the flexion phase of the step, Walshe considers contraction of the hamstring muscle, which occurs synchronously with the toe response, as the "minimal motor response" of the movement of defense.

Any one of these theories is open to the objection that it does not explain the various movements that accompany dorsiflexion of the big toe, such as the fanning out and plantar flexion of the other toes, eversion of the foot and especially contraction of the quadriceps cruris, an extensor of the leg, and contraction of the gluteus maximus, a pure extensor of the thigh. That there is synchronously with the Babinski toe response contraction of the quadriceps cruris has been noted by Cohn,[8] Jacobsohn and Caro,[9] Erwin Loewy[10] and myself.

Fig. 1.—Apparatus for recording the movements in the segments of the limbs

But I have also noted that there is simultaneously an extension of the thigh on the hip. The contraction of the gluteus maximus producing this movement can be frequently observed when the toe sign is obtained while the subject is in the prone position. This is shown graphically in Figures 4 and 5.

To obtain these graphic records, I employed this method: A piece of cord attached to the big toe passed under a pulley to a writing lever in contact with the drum of a kymograph. Dorsiflexion of the big toe was thus recorded by an upstroke of the lever. To record the other movements in the limb I employed the apparatus shown in Figure 1.

8. Cohn, T.: Neurol. Centralbl. **30**:1107, 1911.
9. Jacobsohn and Caro: Neurol. Centralbl. **31**:420, 1912.
10. Loewy, Erwin: Neurol. Centralbl. **32**:90, 1913.

Fig. 2.—Record of the normal plantar response.

Fig. 3.—Record of the normal plantar response.

Fig. 4.—The Babinski toe response with the accompanying movements in the other segments of the limb.

Fig. 5.—The Babinski toe response with the accompanying movements in the other segments of the limb.

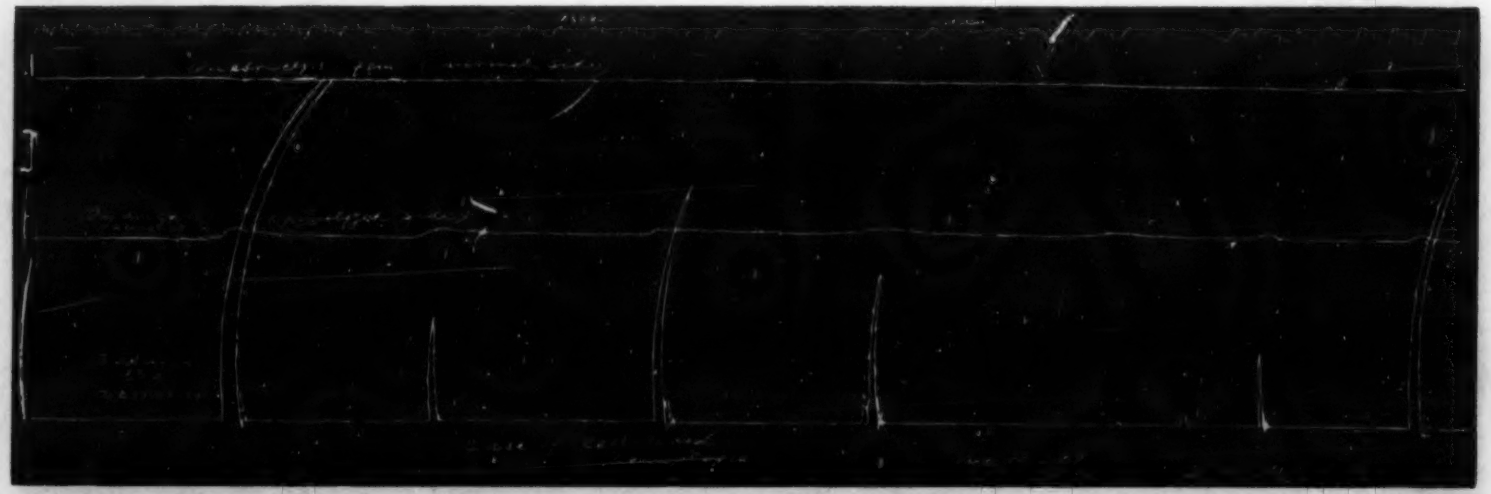

Fig. 6.—The Babinski toe response with the accompanying movements in the other segments of the limb.

Fig. 7.—The Babinski toe response with the accompanying movements in the other segments of the limb.

To the middle of a leather braclet (1) is fastened a metal plate (2). To this plate are riveted a semirigid metal strap over 5 inches long (7) which extends along the surface of the limb in its long axis and a rigid bar (3) rising from its point of attachment so that its end is a couple of inches above the free end of the metal strap. Through the free end of the bar passes a metal rod (4) which rests

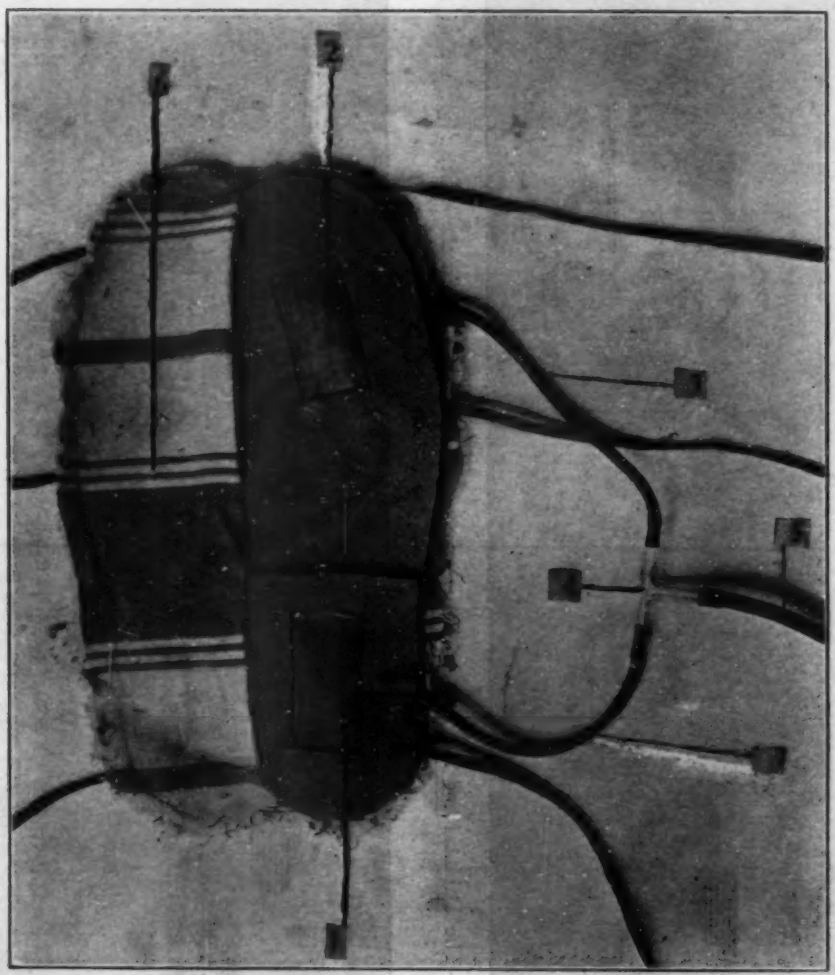

Fig. 8.—Apparatus for recording the movements of the foot in walking.

by a small circular plate (5) on an elastic air chamber (6), fastened to the free end of the metal strap. The rod is secured by a thumb screw where it passes through the bar so that the circular plate is kept firmly in contact with the air chamber. By a rubber tube the air chamber communicates with a Marey's tambour and a kymograph.

Fig. 9.—Record of the various phases of the step.

To record the contraction of the quadriceps cruris and the hamstring muscle, the bracelet encircled the thigh at the lower end and the air chamber rested, respectively, at about the middle of the quadriceps or on the hamstring muscles just below the gluteal fold. To record the contraction of the gluteus maximus the bracelet encircled the thigh at about its middle and the air chamber rested on the lower part of the muscle which extends between its origin at the sacrum and coccyx and its insertion. I recorded the flexion of the knee on the thigh by having the bracelet encircle the thigh at about its middle and the air chamber rest at the lower end of the popliteal space between the two heads of the gastrocnemius. I have graphic records of the movements in the limbs associated with the Babinski toe response from fourteen hemiplegic or paraplegic patients. The results obtained are shown in Figures 4 to 7. The records show that simultaneously with the Babinski toe response there was invariably a contraction of the homolateral quadriceps cruris, gluteus maximus and hamstring muscles. This response was not accompanied by movement in the contralateral thigh (with the exception of slight contraction of the hamstring muscles) when the stimulus was mild. When, however, the stimulus was strong, the contraction of the quadriceps was immediately followed by its relaxation and this was accompanied by contraction of the contralateral quadriceps. In the one case of Friedreich's ataxia I have obtained results in some respects, directly the reverse of the preceding. The records showed that simultaneously with the Babinski toe response there was flexion of the knee with relaxation of the homolateral quadriceps and gluteus maximus (Figs. 11 and 12). This may have been due to the muscular atonia in this disease. In contrast to the response on the paretic side in hemiplegics, was that obtained from the normal limb. When the stimulus was strong the limb underwent a rapid movement of withdrawal, entirely too rapid to be properly recorded; when the stimulus was mild, there was dorsiflexion of the foot, generally immediately followed by plantar flexion of the big toe (the only toe the movements of which were recorded), with no movement in the other segments of the limb (Figs. 2 and 3).

MOVEMENTS OF THE LIMBS IN LOCOMOTION

Since locomotion is the primary and essential function of the lower limbs, I wished to determine whether this combination of movements occurring synchronously with the Babinski sign is reproduced by the limbs at any time during walking. First I considered that the inner metatarsal, as is shown by a number of anatomic facts,[11] forms the

11. Frazer, J. Ernest: Anatomy of the Human Skeleton, London, J. A. Churchill, 1914.

Fig. 10.—Record of the various phases of the step.

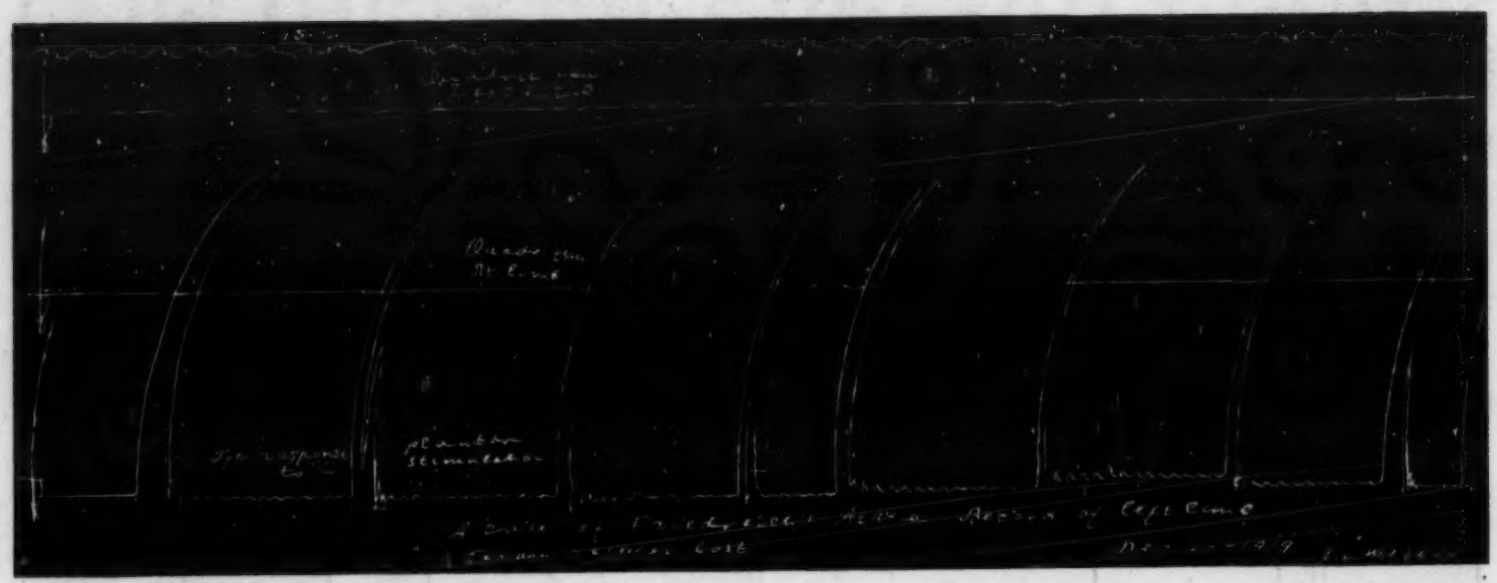

Fig. 11.—The Babinski toe response in a case of Friedreich's ataxia.

medium through which pressure is exerted on the ground during the propulsion period of the step. The outer metatarsus, with its practically immobile articulations, serves chiefly as a guide and point of support for the body and limb while they are rotating anteriorly during this period of the step. Accordingly I fastened to each foot of a normal person a pneumatic pad, such as I employed in my experiments on the locomotor disturbances in disease of the cerebellum (Fig. 8). The anterior air chamber was placed under the inner metatarsal head instead of the center of the ball of the foot as in my previous studies. Records were thus obtained of the movements of the foot while in contact with the ground, as well as of the length of time it is in the air. Movements of the other segments of the limbs during each phase of the step were recorded at the same time by means of the apparatus employed for recording the movements that accompany the Babinski sign. The records obtained are shown in Figures 9 and 10. They show: (1) *a*, the upstroke which initiates the high curve of the record. This corresponds to the moment when the foot is deposited on the ground, coming in contact with it by the heel (Pettigrew [13]). The resulting compression of the air bulb at the heel causes the lever to become elevated. The associated movements in the other segment of the limbs during this phase of the step are: The hip is slightly extending, the knee is slightly flexing, the quadriceps cruris is contracting and the quadriceps of the contralateral limb, which is at this moment exerting pressure on the ground by the toes, (phase *d'*) is relaxing. (2) Immediately following *a*, phase *b* sets in, which is but a continuation of *a*. During this phase (*b*) the whole foot comes in contact with the ground, especially at the heel. The associated movements in the other segments of the limb during this phase are the same as in *a*, except that the extension at the hip is here greatly intensified. The position of the limb is now to a minor degree, similar to that assumed by the limbs at the beginning of a jump, when in order to exert great and rapid pressure on the ground, they are considerably flexed at the knees. The contraction of the quadriceps during these phases, while the knee is slightly flexing (action of the hamstring muscles) is in accord with the view of Winslow,[14] Hunter,[14] Duchenne [15] and

12. Meyers, I. Leon: Locomotor Disturbances in Disease of the Cerebellum, Arch. Neurol. & Psychiat. 2:376 (Oct.) 1919.

13. Pettigrew, J. Bell: Animal Locomotion, New York, Appleton & Co., 1874, pp. 8 and 9, 56-59; Design in Nature, New York, Longmans, Green & Co. 3:1077 and 1122, 1908.

14. Winslow, J. B.: Quoted by C. E. Beevor: The Croonian Lectures, London, Adlard and Son, 1904, p. 52.

15. Duchenne, G. B.: Physiologie des Mouvements, 1867, p. 766.

Fig. 12.—The Babinski toe response in a case of Friedreich's ataxia.

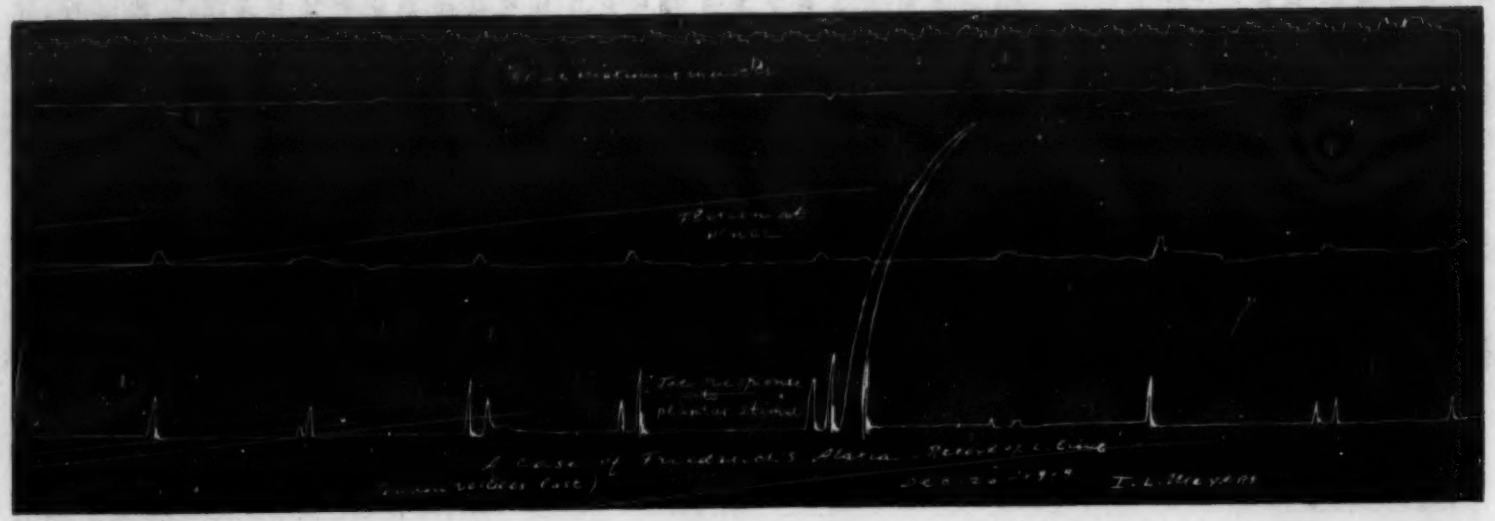

Fig. 13.—The Babinski toe response in a case of Friedreich's ataxia.

others that in movements that are to be accurate and precise, the antagonists act simultaneously with the protagonists. (3) Phase *c*, the pressure on the ground, already at play in the preceding phase, becomes intensified, being now, according to the estimate by Carlet,[16] one-fifth in excess of the body weight. The pressure is now at the ball of the foot exclusively, and in my experiments, at the sesamoid bones of the inner metatarsal, where the air-bulb for the ball of the foot was placed. The associated movements in the other segments of the limbs during this phase are: The homolateral hip is markedly extending, the knee is also markedly extending, the quadriceps cruris is contracting and the quadriceps of the contralateral limb, which is at this time returning to the ground (phase *f'*), is also contracting. (4) Phase *d*, which represents the period when the limb exerts pressure on the ground by the toes only. The pressure on the ground at the toes causes some compression of the air-bulb at the ball of the foot, and is thus recorded by a partial elevation in the curve of the record. During this phase the homolateral hip is markedly extended, the knee is markedly flexed, the quadriceps is relaxed and the quadriceps in the contralateral limb, at this time in phases *a'* and *b'*, is contracted. *d* terminates the period during which the foot is in contact with the ground, and is followed by (5) *e*, which corresponds to the time during which the foot is in the air and advancing. The movements in the other segments of the limbs during phase *e* are: The hip is flexing, the knee continues to be in flexion, the quadriceps is relaxing and the quadriceps of the contralateral limb, at this time in phase *b'*, is contracting. (6) *e* is followed by phase *f*, when the limb is returning to the ground. During this phase the homolateral hip is slightly extending, the knee is extending, the quadriceps is contracting and the quadriceps of the other limb, at this time in phase *c'*, and exerting pressure on the ground by the ball of the foot, is also contracting. It will be seen thus that a complete step is composed of six different and distinct phases, four of these phases forming the propulsion period of the step and two more the period when the foot is in the air.

On looking now at the phases of the step for the combination of movements, which is observed to occur synchronously with the Babinski sign, we find that it is reproduced in phases *a* and *b*, when the body is exerting pressure on the ground mainly at the heel. During this phase of the step, the following movements take place: The big toe becomes dorsiflexed on the foot; the other toes, as shown by Pettigrew,[13] spread out and generally undergo plantar flexion; the whole foot from the direction of movement of the body and limb, which is not only forward, but as shown by Pettigrew and Morey,

16. Carlet, G.: Ann. d. sc. naturelles, **16**:5, 1872.

also horizontally outward, becomes everted; the knee is slightly flexed; the quadriceps cruris and the gluteus maximus are in contraction, and the quadriceps of the contralateral limb, at this time exerting pressure on the ground by the toes (phase *d'*), is relaxed.

THE PHYSIOLOGIC SIGNIFICANCE OF THE BABINSKI TOE RESPONSE

The following theory thus suggests itself. It may be assumed (Marie and Foix) that the function of progression is represented in the spinal cord (l' automotisme medullaire); that normally the movements of progression are kept more or less in check by inhibitory influences of the cerebrum. When, however, inhibition from the cerebrum is cut off as a result of disease of the corticospinal tracts, the spinal centers of progression reassert themselves whenever a stimulus reaches them from the limbs, and the stimulated limb or both limbs execute the stepping reflex in a minor degree. The stepping reflex exhibits itself in the Babinski toe response which represents phase *b*, the first and most important phase in the propulsion period of the step. Phase *b* is the only manifestation of the reflex when the stimulus is mild. When the stimulus is intense, the stepping reflex reaches a much wider extent and passes through, to a minor degree, also phase *d*. The quadriceps cruris of this limb, in contraction in phase *b*, thus becomes relaxed, and there is usually at the same time a movement of plantar flexion of the big toe following the original dorsiflexion. A pronounced reflex of this type affects also the contralateral limb, and, while the stimulated limb is in phase *d*, the quadriceps of the former limb contracts. The most striking features of the Babinski toe response are, the dorsiflexion of the big toe, the spreading out of the lesser toes and the eversion of the foot. This is probably due to the close proximity of the muscles involved in these movements to the point of application of the stimulus (the plantar surface of the foot). If, however, the reflexogenous zone (of the Babinski toe response) extends over the entire limb, (and occasionally also the lower part of the abdomen) the stimulus when applied to the upper part of the limb may, as noted by Marie and Foix, produce extension of the limb (marked action of the gluteus maximus and the quadriceps cruris) and no dorsiflexion of the big toe. The effect of the stimulus is in this instance, too, produced chiefly on the muscles close to the point of stimulus and not on the parts distal to it.

This theory of the Babinski sign explains also the so-called crossed plantar response, frequently observed in paraplegia and occasionally in hemiplegia, which consists of plantar flexion of all the toes with extension of the foot. The phenomenon has been studied and inter-

preted in various ways by Byron Bramwell,[17] Knapp,[4] Fairbanks[18] and others. If the Babinski toe response represents the propelling period of the step that occurs in phase *b*, the contralateral limb at this time being in phase *d'*, the effect of the stimulus to one limb, when the irritability of the nervous system is so great that the reflex affects the other limb also, must be that of plantar flexion of all the toes in the latter limb, the foot being at the same time fully extended at the ankle. Frequent absence of the crossed plantar response may be accounted for by the remoteness of the toes in the nonstimulated limb from the point of application of the stimulus.

This theory may also explain why the Babinski toe response, while obtainable in some conditions by a stimulus applied to any part of the limb and even the lower part of the abdomen, is obtained with such marked facility by pressure at certain foci in the foot and leg. These "receptive foci" are, I believe, the heel, the ball of the foot and the area under the outer malleolus. It will be noted that pressure at the heel and the ball of the foot reproduces the condition which is associated with dorsiflexion of the big toe in walking, and that pressure under the outer malleolus produces an effect on the peroneus longus, the existence of which is, as stated by Huxley,[19] absolutely distinctive of the human foot, a muscle that is essentially an organ of locomotion.

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17. Bramwell, Byron: Rev. Neurol. & Psychiat. **1**:795, 1903; ibid. **9**:49, 1911.
18. Fairbanks, A. W.: Rev. Neurol. & Psychiat. **9**:225, 1911.
19. Huxley, T. H.: Man's Place in Nature, New York. O. Appleton & Co., 1896, p. 124.

THE MOTOFACIENT AND NONMOTOFACIENT CYCLES IN ELEVATION OF THE HUMERUS*

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NEW YORK

The generally accepted view that in raising the humerus to a straight angle, the deltoid elevates the arm approximately to a right angle, after which elevation is completed by scapular rotation, is not supported by our investigation. The deltoid alone is unable to raise the humerus beyond 60 degrees while elevation from this to approximately 115 degrees involves scapular rotation, after which the deltoid again becomes active and completes the elevation.

Inspection and palpation or electrical stimulation of the deltoid will verify the active rôle it plays in elevation of the arm from 115 degrees to a straight angle. The deltoid acts first as an abductor, abducting the arm approximately 60 degrees, and then after elevation to 115 degrees it becomes an adductor, thereby raising the arm through the final arc to 180 degrees.

The origin and insertion of the deltoid are so placed that it may serve this dual function, providing the arm is considered in its position in different stages of elevation. Elevation of the arm has been difficult to interpret, since any movement involves the complex musculature of the shoulder girdle, whose component muscles have wide and diverse origins, and are capable of independent contraction in different parts of the same muscle, subserving at different times different functions.

In paralyses in which the scapula cannot participate in elevation, the deltoid alone is unable to bring the humerus above 60 or 65 degrees. Normally in any abduction beyond 6 degrees, rotation of the scapula takes place and may be made apparent, by fixing the inferior angle of the scapula between the fingers during the act of abduction, when it will be noted that as soon as the humerus reaches an angle of approximately 60 degrees rotation of the scapula occurs. When the humerus is abducted to 75 degrees, the scapula has rotated 20 degrees—thus of 75 degrees abduction, 20 degrees are the result of scapular rotation and only 55 are due to humerus abduction from the scapula. This may be seen by comparing the accompanying illustrations[1] showing the normal relation of the scapula to the humerus

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* Read before the New York Neurological Society, May, 1920.

1. I wish to express my thanks to Dr. Scholz who kindly took the roentgenograms from which these tracings were made.

and to a perpendicular through the shoulder joint. These tracings were made from a series of roentgenograms taken of the writer under constant conditions. The tube was 42 cm. from the plate, the body erect, and the scapula unsupported. (In lying down, the scapula may receive support due to the weight of the body, etc., and its movements be somewhat altered.)

The arc of elevation should be broken into its functional units rather than into the mathematical components of a right angle and a straight angle. These are not functional units and do not admit of interpreting the relation of scapular rotation in the different cycles of elevation of the humerus. The roentgen ray was used by Steinhausen [2] in studying the movements of the scapula, but he divided the movements into various stages from a mathematical standpoint, rather than from physiologic and anatomic considerations, and thereby failed to realize the proportionate relation of scapular rotation to elevation of the shoulder.

THREE PHYSIOLOGIC CYCLES IN ELEVATION OF THE ARM

We have found the functional units in the elevation of the humerus to be: first, elevation of the arm in which there is little or no rotation of the scapula (60 degrees); second, elevation during which the major portion of scapular rotation takes place (60 to 115 degrees); third, the final arc of elevation in which scapular rotation has relatively ceased and elevation of the humerus is continued to a straight angle (115 to 180 degrees).

First Stage: From a study of Figure 1, it will be noted that the humerus is abducted 60 or 65 degrees, and the scapula rotated 5 or 6 degrees. Of this movement, approximately 55 degrees are due to the action of the deltoid and supraspinatus.

Second Stage: In further abduction and elevation from 60 to 115 degrees, the scapula is rotated an additional 35 degrees. Thus, in this arc of 55 degrees, 35 degrees is accomplished by scapular action and only 20 degrees by the deltoid.

Third Stage: The elevation from 115 to 180 degrees is accompanied by only 10 degrees of scapular rotation. In this arc of 65 degrees the scapula accomplished but 10 degrees, the remainder being unaccounted for by the older view, which attributes all of this final arc, as well as part of the second, to rotation of the scapula.

First Cycle.—During the first stage, the arm is abducted to 60 degrees by the deltoid and also to a lesser extent by the supraspinatus, and is accompanied by a slight rotation of the scapula, more particu-

2. Steinhausen: Beitrage zur Lehre von dem Mechanismus der Bewegungen des Schultergürtels, Arch. f. Anat. u. Physiol. 1899, p. 403.

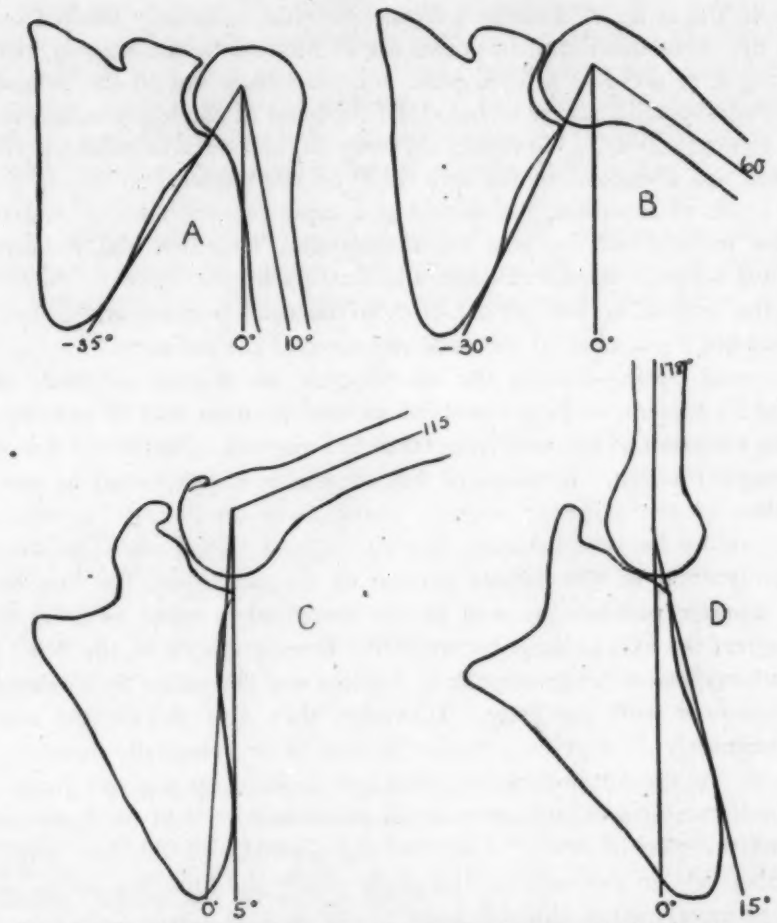

Fig. 1.—Tracings from roentgenograms showing the relation of the scapula to the humerus and to a perpendicular through the shoulder joint in various phases of elevation of the humerus. A line has been carried in each along the axillary border and through the inferior angle of the glenoid cavity. The line through the humerus is carried through the center of its longitudinal axis. *A*, the normal relation of the scapula to the humerus. The scapula forms an angle approximately 35 degrees with a perpendicular, and the humerus 10 degrees. The humerus may be brought to the perpendicular without any alteration in the relative position of the scapula. *B*, the humerus has been raised 60 degrees, during which the scapula has rotated approximately 5 degrees. This rotation of the scapula is rather in the sense of fixation than a participation in elevation of the humerus. *C*, the humerus has been raised to 115 degrees, during which the scapula has rotated 35 degrees from its position in *B*, and 40 degrees from its position in *A*. In elevation from 60 to 115 degrees, the scapula has rotated 35 degrees or 70 per cent. of its total rotation. *D*, the humerus has been raised practically to a straight angle, during which the scapula has rotated an additional 10 degrees from its position in *C*, or a total of 50 degrees from its original position in *A*. In this last cycle of elevation, the scapula has rotated 20 per cent. of its total.

larly in the sense of fixation. Duchenne [3] has accurately pointed out that the rhomboideii and trapezius act as fixators of the scapula, permitting it to serve as a fixed point for the attachment of the deltoid. The supraspinatus serves to help hold the head of the humerus against the glenoid cavity. However, the only action he attributed to the deltoid was abduction of the arm to 90 or 100 degrees. Thus, in the first cycle of elevation, the deltoid and supraspinatus may be said to to the motofacient factors; the rhomboideii, trapezius and serratus magnus serve as nonmotofacient and fixators of the scapula. In this arc, the scapula rotates 10 per cent. of its total rotation and accomplishes but 8 per cent. of the total elevation of the humerus.

Second Cycle.—During the second cycle the scapula subtends an arc of 35 degrees, or 70 per cent. of its total rotation, and 65 per cent. of the elevation of the arm from 60 to 115 degrees. This is the arc of its major rotation. Rotation of the scapula is accomplished by contraction of the serratus magnus, particularly the lower digitations which rotate the inferior angle laterad. This action is also furthered by contraction of the middle portion of the trapezius; the inferior and superior portions, as well as the rhomboideii, serve as fixators, and direct the axis of scapular rotation. During this cycle, the deltoid and supraspinatus act principally as fixators and determine the angle of the humerus with the body. However, they also elevate the arm approximately 20 degrees, yet may be said to be essentially nonmotofacient. In the latter function, they are assisted by the tres major, pectoralis major and latissimus dorsi, which help to hold the humerus against the glenoid cavity as did the supraspinatus in the first stage, and also help in determining the angle which the humerus forms in the horizontal plane with the body.

Third Cycle.—In the third cycle, from 115 to 180 degrees, the scapula participates but 10 degrees, or the scapula rotates 20 per cent. of its total rotation and accomplishes 15 per cent. of the total elevation in this stage. Thus 55 degrees of elevation are accomplished without the assistance of the scapula. This last cycle is performed principally by the deltoid and supraspinatus, more specially the clavicular head of the deltoid. However, this depends on the horizontal angle which the humerus forms with the trunk. The more ventrad the humerus, the more stress is thrown on the clavicular head of the deltoid, and the further dorsad the humerus, the more the median fibers participate. The posterior fibers serve as guy ropes, together with the teres major and latissimus dorsi, to determine the angle with which elevation is accomplished.

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3. Duchenne: Physiologie des Movements, 1867, J. B. Balliere et Fils.

With the arm at 115 degrees, the supraspinatus and deltoid are ideally placed to exert considerable force in the direction of elevation. It might be said that the supraspinatus muscle and the deltoid now complete the elevation by adduction. Thus the deltoid has again become motofacient, and the scapular muscles essentially nonmotofacient, serving primarily to fix the scapula, though they participate slightly in elevation.

In raising the humerus from zero to 180 degrees, the scapula rotates a total of 50 degrees; the remaining 130 degrees of elevation are humerus action, due chiefly to the supraspinatus and deltoid, more

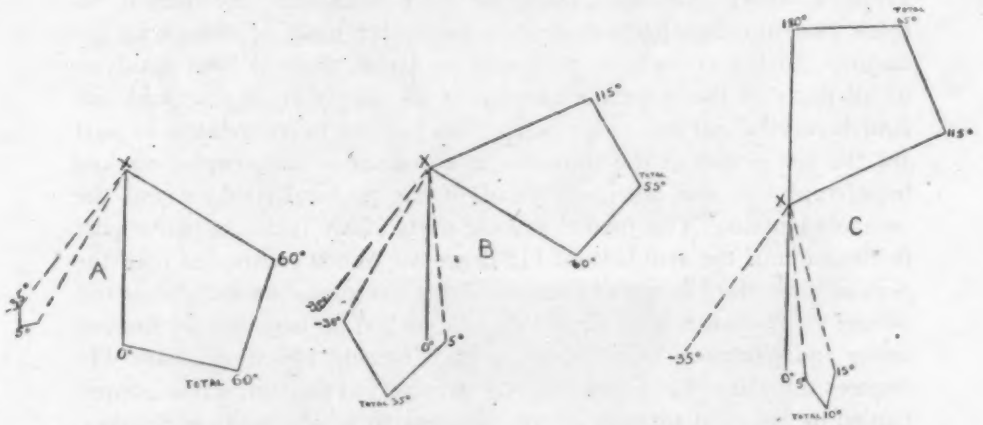


Fig. 2.—Diagrams taken from Figure 1 to illustrate the relationship of rotation of the scapula to elevation of the humerus. *A*, during elevation of the humerus to 60 degrees, the scapula has rotated 5 degrees; 55 degrees represents elevation of the humerus alone. Thus 8 per cent. of elevation is due to scapular rotation, and the scapula rotates 10 per cent. of its total. *B*, elevation of the humerus from 60 to 115 degrees, during which the scapula has rotated but 35 degrees; 20 degrees is the elevation of the humerus alone. The scapula rotates in this cycle 70 per cent. of its total. *C*, elevation of the humerus 65 degrees from 115 to 180, during which the scapula has rotated 10 degrees; 55 degrees is the elevation of the humerus alone and unaccounted for by any rotation of the scapula. In this last cycle of elevation, 15 per cent. is due to scapular rotation. The scapula rotates 20 per cent. of its total.

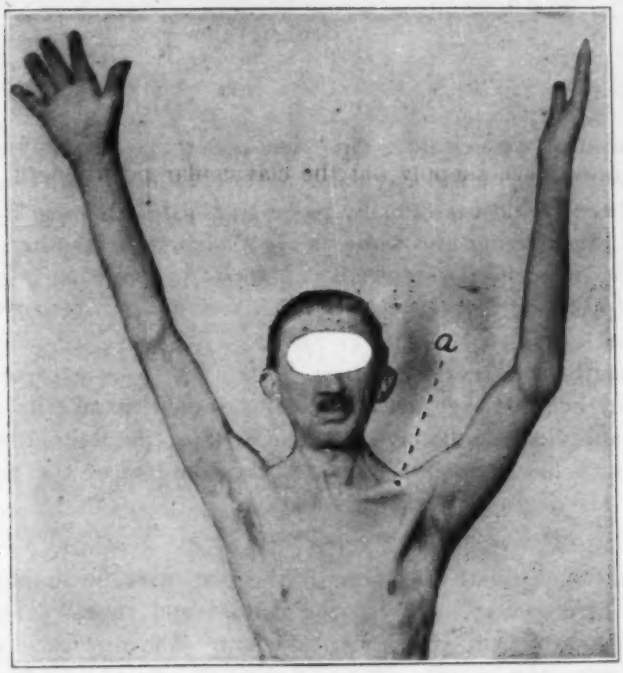
particularly the clavicular head of the latter, especially in the final cycle of elevation.

Thus alternating cycles form the functional units in the different stages of elevation, in the first of which the deltoid is motofacient and the scapular muscles are nonmotofacient; in the second, the scapular muscles are motofacient, and the deltoid, supraspinatus, teres major, pectoralis major and latissimus dorsi are nonmotofacient; while in the third cycle, the deltoid and supraspinatus again become motofacient and complete the act of elevation.

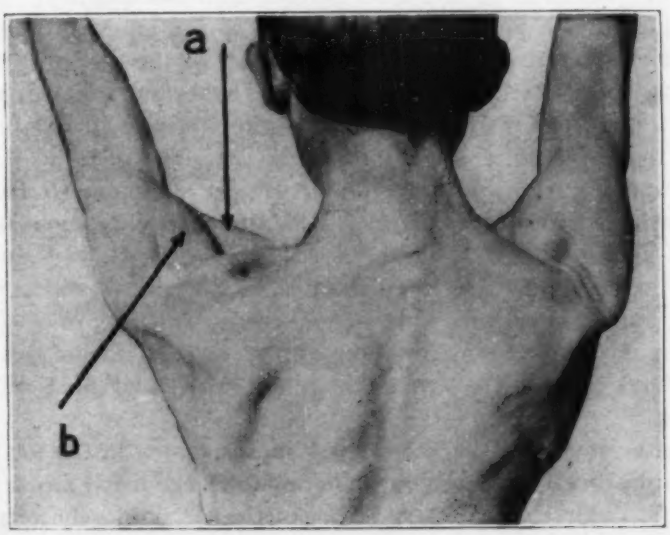
Action of Clavicular Head of Pectoralis Major and Coracobrachialis in Final Stage of Elevation.—Two muscles, whose action in elevation has been ignored, may serve as supplementary factors of no small importance in accomplishing the final stage of elevation. They are the clavicular head of the pectoralis major and the coracobrachialis. These muscles are brought into play particularly when great force in elevation is demanded, or when there is some impairment of the function of the deltoid, or of the muscles which fix and rotate the scapula. The accompanying illustrations taken from a case in which the trapezius was totally paralyzed, due to the severance of the spinal accessory nerve a short distance below its exit, illustrate the action of these two muscles, particularly the clavicular head of the pectoralis major. So far as we have been able to detect, there is total paralysis of all parts of the trapezius in spite of its supply from the third and fourth cervical nerves. This patient has learned to compensate in part for the lost action of the trapezius in elevation of the arm by marked hypertrophy of the clavicular head of the pectoralis major and the coracobrachialis. The former muscle particularly takes an active part in elevation of the arm beyond 115 degrees. When contrasted with the normal side, the clavicular portion of the pectoralis major stands out almost as a separate muscle and may be picked up between the fingers, being approximately 3 to 4 cm. thick. During elevation above 115 degrees the clavicular portion shows definite visible contraction accompanied by outward rotation of the humerus, in which position elevation is more readily accomplished. A front and back view, as illustrated in Figure 3, show the contraction of this muscle in the act of elevation, thus indicating its active rôle in the accomplishment of this movement.

Normally, this function of the clavicular head of the pectoralis major may be brought out by forcible resistance to elevation or by sudden attempts to lower the arm from elevation, particularly if the patient is able to oppose suddenly and vigorously each of these movements. The clavicular portion of the pectoralis major will be seen to take active part in such forcible elevation above 115 degrees, or oppose efforts to lower the arm to 115 degrees.

In this connection, it is interesting to note that phylogenetically the clavicular head of the pectoralis major is present only as such in the higher forms, particularly in the chimpanzee and the gibbon, while in all other primates as well as the lower mammals, it is wanting. In them, the pectoralis major arises from the manubrium and does not reach to the clavicle. However, in those animals in which there is no clavicular head of the pectoralis major, the clavicular portion of the deltoid extends mesially on the clavicle up to the origin of the sternocleidomastoideus. The clavicular portion of the pectoralis major may be considered as a migration of the innermost muscular fibers of the



A



B

Fig. 3.—Photographs of patient showing marked hypertrophy of the clavicular head of the pectoralis major. The trapezius has been totally paralyzed for ten years. *A*, on the left side, the clavicular portion of the pectoralis major shows marked hypertrophy and stands out almost as a separate muscle. An interval can be seen between the clavicular head and the sternal portion. In movements with the arm at the side, the pectoralis major contracts as a whole and shows no anomaly. The separation of the clavicular and sternal portions takes place only in elevation of the arm above 115 degrees. Note that the arm is held in external rotation. *B*, Posterior view of the same patient showing the active rôle of the clavicular portion of the pectoralis major in elevation of the arm from above 115 degrees. Note also on both sides the contraction of the deltoid, particularly the clavicular head, showing the active participation of this muscle in the elevation of the humerus—a function generally ignored. *a*, clavicular head, pectoralis major; *b*, clavicular head of the deltoid.

clavicular head of the deltoid. This view is perhaps further sustained by the nerve supply of the clavicular portion of the pectoralis major which has been found occasionally to be supplied by a twig from the circumflex, thus having the same nerve distribution as the deltoid. Furthermore, normally it receives its innervation from that portion of the external anterior thoracic nerve which receives its fibers from the fifth segment.

Consequently, it is not surprising, but rather to be expected, that the clavicular head of the pectoralis major should resume its former association and along with the clavicular head of the deltoid serve in part, at least, a like function, namely, as an elevator of the humerus from above 115 degrees.

CONCLUSIONS

1. The mechanism of elevation of the arm may be divided into three physiologic cycles in which the deltoid and supraspinatus are alternatingly motofacient and nonmotofacient. The first cycle of elevation is accomplished by the action of the deltoid and supraspinatus, raising the humerus to about 60 degrees, and accompanied by slight rotation of the scapula; the second cycle is accomplished principally by rotation of the scapula due to the action of the serratus magnus, trapezius, rhomboideii, and levator angulii scapulae, which raises the arm to approximately 115 degrees, and is accompanied by slight elevation of the humerus; and the third cycle is accomplished by elevation of the humerus from 115 degrees to a straight angle due to the action of the deltoid and supraspinatus in which there is only slight rotation of the scapula.

2. The older view that the deltoid raises the arm to approximately a right angle, after which its action ceases and the movement of elevation is completed by rotation of the scapula, is not sustained. The deltoid elevates the arm both in the initial and final stages of elevation.

3. Associated with the deltoid and supraspinatus in the final stage of elevation are the coracobrachialis and the clavicular head of the pectoralis major. The latter, phylogenetically, is closely related to the deltoid and in its development may be considered as a migration of the deltoid fibers. Hence, it is not surprising that this muscle should resume its former association in function and assist in elevation of the humerus from above 115 degrees.

471 Park Avenue.

News and Comment

ANNUAL MEETING OF THE AMERICAN MEDICO-PSYCHOLOGICAL ASSOCIATION

At the seventy-sixth annual meeting of the American Medico-Psychological Association the following officers were elected: president, Dr. Owen Copp, Philadelphia; vice president, Dr. Sanger Brown, Kenilworth, Ill.; secretary-treasurer, Dr. H. W. Mitchell, Warren, Pa. Dr. George M. Kline of the Massachusetts State Commission of Mental Diseases is chairman of the entertainment committee, and Dr. Stanley Abbott is chairman of the program committee.

The next annual meeting is to be held at Hotel Somerset, Commonwealth Avenue, Boston, May 31, June 1, 2 and 3, 1921.

In addition to the annual meeting the association will, during the coming year, take up the question of revision of the constitution. Among other changes suggested is change of name of the association from the American Medico-Psychological Association to the American Psychiatric Association.

Abstracts from Current Literature

THE SPIROCHETE OF GENERAL PARALYSIS. LEVADITI and MARIA, Ann. de l'Inst. Pasteur **33**:741, 1919.

This study on the spirochete of general paralysis represents the continuation of experimental work interrupted by the war.

In 1913 (Compt. rend. Acad. d. sc. **157**:864, 1913; ibid. **158**:1595, 1914) the authors, in collaboration with Danulesco, succeeded in transmitting spirochetes from a case of paresis to the rabbit, and in producing typical lesions. Several passages were made and comparative biologic and pathologic studies were made of this virus and the virus of human syphilis, cutaneous and visceral. The study showed such manifest differences between the two parasites that the conclusion was inevitable that the spirochete of general paralysis must be regarded as a distinct, neurotropic variety of *Spirochaeta pallida*, and that the pathologic agent of syphilis is present in the circulation of the paretic.

A complete review of the literature is made regarding the demonstration of the spirochete in the cerebral cortex, the blood and the spinal fluid in cases of paresis. The results of all these studies show that the spirochete is present in the cerebral cortex, blood, and spinal fluid in all cases of paresis, irrespective of the clinical manifestations, progress and severity of the disease; and that the presence of spirochetes can be demonstrated either by microscopic examination (brain) or by inoculation experiments. Although the positive results of these experiments are by no means constant, they show a remarkable increase with the increased number of experiments and the abandonment of postmortem studies for observation in vivo and the use of the ultramicroscope.

The demonstration of spirochetes in the cerebral cortex of paretics is almost a constant finding, but is less frequent in the blood and spinal fluid. In the blood and spinal fluid the presence of the parasite is probably intermittent and transitory.

Clinical observations are presented which suggest the existence of a neurotropic strain of *Spirochaeta pallida*. These observations are: 1. The fact that early syphilitic manifestations in paretics and tabetics are slight, transient or absent, and the tertiary manifestations in the skin and viscera exceedingly rare. Fournier's observations on 243 cases of severe syphilis are quoted. These cases were observed by Fournier for a number of years and not one developed paresis or tabes. 2. The rarity of paresis among natives of tropical countries where syphilis occurs in a severe form and is treated insufficiently or not at all. 3. The existence of conjugal or familial neurosyphilis; although the authors state that the number of such instances are not numerous considering the frequency of tabes and paresis. A number of the reported cases of conjugal neurosyphilis are presented.

Two strains of spirochetes were studied. One was obtained by Touffi from a patient with cutaneous syphilis, the other by the authors from the blood of a paretic. The former is termed dermotropic virus and the latter neurotropic virus.

The viruses show a difference in their incubation period, in the character of the lesions, the progress and virulence of the disease. The incubation period of the neurotropic virus is much longer (55, 60, 77, 94 days) than the dermotropic virus (43, 46, 49, 60, 77 days).

Character of Lesions: The dermatropic virus produces a papulosquamous erosion. Microscopic examination reveals erosion and desquamatous superficial ulceration of the epidermis, vascular lesions without periarteritis or endarteritis of the dermis, and slight infiltration. There is no conjunctival neoformation. The spirochete is found in the epithelial layer.

The neurotropic virus produces a papulosquamous erosion. Microscopic examination reveals erosion, desquamation and superficial ulceration of the epidermis, vascular lesions without periarteritis or endarteritis of the dermis, and slight infiltration. There is no conjunctival neoformation. The spirochete is found in the epithelial layer.

The dermotropic virus produces an indurated chancre. Microscopic examination reveals deep ulceration of the epidermis, peri-arteritis and endarteritis of the dermis, and intense infiltration. There is intense conjunctival neoformation. The spirochete is found in the deep layers.

Virulence: The two viruses show a striking difference in virulence. The dermotropic virus retains its virulence for the lower apes and the anthropoid apes even after many passages through the rabbit, tested during the years 1908-1914. Through the accidental inoculation of one of the assistants in the experiments, the authors were able to demonstrate that it continues to be pathogenic for the human subject as well, the local lesions, however, in the latter were mild, no secondary symptoms developed and the Wassermann reaction became positive long after the local lesion appeared.

The virulence of the neurotropic virus was studied in two cases of paresis, and it was found that this virus, which was pathogenic for the rabbit and apes and the human subject, was avirulent for the paretics. On the other hand, the neurotropic virus is avirulent for the ape, if inoculated after a single passage through the rabbit; similar results were obtained from experiments on the human subject.

Crossed inoculation experiments showed that rabbits which had recovered from infection with dermatropic virus and were vaccinated again this virus developed, when inoculated with neurotropic virus, the characteristic experimental papulosquamous lesion. The inverse experiment was positive.

The conclusion, therefore, appears justified that there is a biologic difference between the two parasites. Experiments conducted to justify this conclusion showed that rabbits after presenting the local lesion provoked by dermatropic or neurotropic virus, acquire a refractory condition with regard to a homologous spirochete, and retain their receptivity toward a heterogeneous virus. The dissemblance between the two viruses is at least as marked as that existing between the spirilla of Dutton and that of Obermayer, the pathogenic agents of African and European relapsing fever.

The question whether or not syphilitics destined to develop tabes or general paralysis are contaminated at the onset of their syphilis by a special spirochete, or whether this special type, owing to its residence in the central nervous system, is created as the result of a progressive adaptability, is one that is not easily determined. While some of the observations cited seem to indicate an original contamination, the authors believe that the two hypotheses are not only perfectly compatible, but rather tend to confirm the theory of a neurotropic virus. Among the numerous syphilitic germs, morphologically similar, but differing in virulence, which transmit syphilis in civilized countries as the result of old infection, there are some which are distingushed by a special aptitude to localize in the central nervous system. This does not imply that they are incapable of localizing in the skin or in certain viscera or in the cerebrospinal fluid. Otherwise we would not be able to understand their abiltiy

to be transmitted from one individual to another by the cutaneous, mucous or circulatory route (hereditary). But they do seem from the very start to have a weak reaction on the skin and the mucous surfaces, as is evidenced by the transitory symptoms and their mildness as seen in the future paralytics or tabetics.

This neurotropic spirochete, however, is very early transformed into one whose affinity for the brain and spinal cord becomes more and more marked and almost exclusive, in that the parasites thrive in contact with the cerebrospinal substance. And it is precisely this type of spirochete which we have been studying and which we have compared with the dermatropic virus.

It also seems that the difference between the two viruses is not so great at the time of the initial contamination as it is when paresis or tabes is fully developed. In other words, the spirochete treponema that later becomes neurotropic protects the human subject against cutaneous and mucous syphilis, while the parasite already neurotropic does not immunize the rabbit against the dermatropic virus. In short, the budding of so-called parasyphilis appears to be due to a special kind of contamination by a type of spirochete with a more or less neurotropic affinity. This neurotropic character may be so definite that the lesions in the cerebral cortex and the spinal cord have nothing in common with those generally considered by pathologists as characteristic of syphilis. In fact, many who have studied the histology of paresis emphasize the lack of resemblance between the existing syphilitic change proper (endarteritis, gumma, meningitis, etc.) and those of paresis.

The question has assumed greater importance in recent years. The war and certain occupational diseases seem to show that nervous syphilis, apparently latent, may become active and progress rapidly into a galloping general paralysis. Our theory would seem to indicate that the lowered resistance of the cerebrospinal centers favor local pullulation of a latent neurotropic virus.

The authors realize the difficulties and the defects of the study. Many questions remain unsolved, prominent among them the inefficacy of various antisyphilitic treatments in general paralysis and tabes. This will be considered in a future publication. In the meanwhile they repeat their belief, expressed in 1914, that this inefficacy is not due to the resistance to arsenic of the neurotropic virus, since rabbits infected with that virus recovered just as well as animals infected with dermatropic virus (of Truffi), after intravenous injections of arsenobenzol.

KLAUDER, Philadelphia.

ADVERTENCIA DE LA REDACCION (EDITORIAL NOTE). Arch. de neurobiol., psic., fisiol., histol., neurol. y psiquiat. **1**:1, 1920.

The introductory word by the editor is of interest as it throws some light on the conditions under which scientific work is done in Spain at present.

The editor calls attention to the recent progress in neurology and psychiatry in Spain which is due to the scientific activity of a studious set of young men formed largely by association with Cajal and Simarro, and by the work and study in the clinics and laboratories of those nations most advanced in science. The editor notes that this progressive movement has taken place outside of the university which "does not consider these activities worthy of a chair."

"This journal, therefore, is founded to consolidate and organize this scientific movement to receive the work of Spanish masters and of the new generations of investigators and to make the work of Spanish students known in progressive lands."

ATAQUES EPILEPTICOS EN LA ESQUIZOFRENIA (EPILEPTIC FITS IN SCHIZOPHRENIA). José M. Sacristán, Ibid., p. 17.

The author offers nothing new or original in regard to epileptic convulsions in schizophrenia, but he reviews the latest articles on the subject giving a good bibliography and presents his own two cases as confirmatory of the Kraepelin-Bleuler theories: the metabolistic-toxic origin of the convulsions in schizophrenia.

LAS NEURITIS POSTGRIPALES (POSTINFLUENZAL NEURITIS). Jose M. de Villaverde, Ibid., p. 23.

This article contains a careful and detailed consideration of postinfluenzal neuritis and with right emphasizes the necessity of distinguishing between paralyses occurring from neuritis and those caused by nuclear foci. The large number of third and sixth nerve involvement, without any lethargic symptoms, is interesting.

UN CASO DE TUMOR DEL CUERPO CALLOSO SIN DISPRAXIA IZQUIERDA (A CASE OF TUMOR OF THE CORPUS CALLOSUM WITHOUT LEFT DYSPRAXIA). Gounzalo R. Lafora, Ibid., p. 45.

The comparative rarity of tumors of the corpus callosum renders every well studied case of interest especially if, as in the case presented, some symptom or symptoms are unexpected. The author has made a careful analysis of his case and it is presented with an outline of the work done by Liepmann, Déjerine and others.

ALGUNOS OBSERVACIONES SOBRE LA HISTOGENESIS DE LA NEUROGLIA EN EL BULBO OLFATIVO (SOME OBSERVATIONS ON THE HISTOGENESIS OF THE NEUROGLIA IN THE OLFACTORY BULB). F. de Castro, Ibid., p. 49.

The author treats briefly the dual theory of the origin of the neuroglia: that it is produced in part by the ectodermic tissues and in part by the mesodermic, and the unitarian or ectodermal theory of the origin of glial tissues, and from his own research ranges himself on the side of the ectodermal theory.

His work was done on the olfactory bulb of the dog and cat at birth, and at 10, 20 and 40 days. He made use of Cajal's gold sublimate method and the same master's uranium-formol method. His conclusions are:

1. In the olfactory bulb of the new-born dog and cat all the phases of the evolution of the primitive epithelial cell can be followed up to the production of the glial cell, thus confirming Cajal's theory that the neuroglial cells are the product of epithelial cells or spongioblasts adapted to the environment.

2. In the molecular zone twin glial cells or groups are numerous, showing the activity of the proliferation of the glia at this period of its development.

3. The neuroglia of animals of a few days' existence is polarized in all the different layers and maintains its orientation toward the bulbar surface.

4. When the genuine epithelial cell is in relation to the ventricular cavity it shows a vigorous internal expansion, while the external prolongation is fine, free in its course and terminates in a point without ever being implanted on the pia mater.

5. In all the cells of the ependymal wall the centrosome, Golgi's reticulum and the mitochondrial granules are lodged in the interior of the cell or looking toward the ventricular cavity.

6. As soon as the epithelial element begins to detach itself and leave the ventricle, the crest emigrates to the surface, its exterior becomes netlike or branched and insertion into the vessels takes place at the free extremities. In the meantime the internal expansion becomes slender and finally atrophic and disappears.

7. In cells recently detached the centrosome and apparatus of Golgi have changed their position in the cell, both being in the exterior segment of the cell or in the direction of the largest peripheral branch.

8. The attraction of the vessels for the neuroglial protoplasma in the formation of glial feet (suckers, chupadores) must be relatively slight or nil. Probably previous trophic exigencies of the neuroglial cell exist in modeling the neuroglial feet.

9. In the evolution of the epithelial cell the phenomenon of retrograde atrophy of the radial stalk and also some of its collaterals is specially noted.

10. The collaterals which arise in the course of the radial stalk take their origin in the most robust side branches (vellosidades) on the stalk referred to and singularly enough in those placed peripherally. The formation of the collaterals is a manifestation of the most advanced period in the evolution of the neuroglia, appearing, probably, preceding the retrograde atrophy of the peripheral expansion, and being a necessary formation in the extreme differentiation of the glial element.

11. Some of the arguments exposed in the preceding conclusions in regard to the epithelial cell of the mammifers may be noted also in the same body in the philogenetic series. The batrachians and reptiles form an exception. In these vertebrates the vascular insertions take place commonly on peripheral capillaries and are established in collaterals coming from the stalk.

NUEVOS METODOS DE ANALISIS DEL LIQUIDO CEFALO-RAQUIDEO (NEW METHODS OF ANALYSIS OF THE CEREBRO-SPINAL FLUID). Gounzalo R. Lafora and Miguel Prados y Such, Ibid., p. 80.

This article on new methods of analysis of the cerebrospinal fluid does not give any methods peculiar to the authors but considers the technic and the value of the mastic reaction as done by Emmanuel and modified by Kafka and the Prussian blue test published by Kirchberg in 1917.

In the majority of their cases the authors found that the mastic reaction coincided with the Lange gold test. The gold reaction from blue to colorless (3 to 5) always gives positive reactions in the mastic, almost always when the curve is low (color 5 colorless) the mastic reaction is positive in four tubes (++++). In median Lange curves (3443321100 or 4433321000) the mastic is positive only in two or three tubes. The minimum limit of Lange's reaction in which a mastic reaction is found corresponds to the curves in which some tube is blue (222321000) or color 3. One exception only was observed to this rule in the case of a probable brain abscess caused by a suppurating mastoid.

The mastic reaction does not answer as a diagnostic means for differentiating general paralysis of the insane from tabes and cerebral syphilis, at least, if used by the Emmanuel method. In optic neuritis the mastic coincides with Lange's in being negative. In nonsyphilitic diseases (epilepsy, dementia praecox, angioneurotic edema) Lange and mastic coincide in negativity. In

the case of supposed brain abscess mastic was positive (++) and Lange almost negative (22110000000).

The authors have found Prussian blue uncertain in its results and altogether inferior to the mastic.

GURD, Ann Arbor, Mich.

ZUR ENTWICKELUNG UND PATHOLOGISCHEN ANATOMIE DER RAUTENPLEXUS (THE EMBRYOLOGY AND THE PATHOLOGIC ANATOMY OF THE CHOROID PLEXUS OF THE FOURTH VENTRICLE). C. VON MONAKOW, Schweizer Arch. f. Neurol. u. Psych. **5**:378, 1919.

The contribution represents the results of a further study of the relation that the choroid plexuses bear to diseases of the central nervous system. In a previous article (*Arch. Neurol. & Psychiat.* **3**:182, 1920) von Monakow and Kitabayashi reported extensive degenerative changes in the choroid plexuses of the hemispheres in severe forms of psychic disturbances, particularly those exhibiting schizophrenic manifestations.

Complete serial sections of the brains of about fifteen human embryos from 1 to 20 cm. in length, representing from the second to the fourth fetal months, and sections from more fully developed fetuses and from the brains of children were prepared. It was noted that the ventricles of the oblongata could be divided into completely separated compartments, one medial and two lateral. These spaces contain the medial and lateral plexuses of this ventricle; they are lined with ependymal cells, are completely separated from each other, and communicate nowhere with the subarachnoid spaces. In a fetus 1 cm. in length there are accordingly no morphologically preformed foramina of Magendie or of Luschka. In one of 2.4 cm. we have as yet no evidence of a choroid plexus. There is noted an accumulation of hematopoietic cells representing the anlage of the plexus; the primitive folds of the lateral plates at this time assume a villous character. The choroid plexus of the fourth ventricle is directed medially from the region of the lateral recess, while that of the lateral diverticulum arises from the region of the corpus restiforme and is directed ventrolaterad. The villi, at this stage, are covered with well differentiated, glandlike cells derived from the ependymal matrix, but are as yet almost entirely free from blood vessels. Even thus early, protoplasmic bodies are liberated from these cells and enter the ventricular spaces. Monakow is of the opinion that these deliver material, through the medium of ventricular fluid, which is necessary for the further tectonic development of the oblongata. In embryos of 3 cm. length the bilateral anlage of the cerebellum is readily discerned; in those of 8 cm. the flocculus shows its appearance. Nowhere do the chambers of the lateral diverticulum and that of the fourth ventricle proper appear to communicate.

In a child of 1 year the anatomic relations are exactly the same as in an adult, save that the blood vessels are smaller and the pericapillary connective tissue more delicate. Monakow insists that serial sections even of the adult brain show that the location of the foramina of Luschka represent merely the place of entry of the larger blood vessels into the plexuses; a preformed communication between the ventricles and the subarachnoid spaces does not exist. Also, in all probability, the lateral diverticulum and the fourth ventricle do not communicate directly. The wall of the diverticulum consists of (*a*) ependymal cells, mostly of a single layer, arranged in the form of pallisades, (*b*) subependymal tissue in which glial elements, small nerve cells, unmedullated nerve fibers, and

molecular substances occur, and (*c*) a dense outer layer which separates these structures from the subarachnoid space.

Monakow sees a great biologic significance in the physiologic-pathologic relation of the tuberculum acusticum, the ventral acustic ganglion, and the lateral walls of the flocculus to the lateral diverticulum. The lateral and caudal portion of the acoustic tubercle, as well as of the ventral ganglion, are covered completely with ependymal cells, permitting direct contact with the villi of the choroid plexus, providing nutritive material in the way of ferments; on the other hand, the medial and anterior portion of the acustic region is located outside of the diverticulum, is free from ependyma but clothed with vascular arachnoid, and is consequently nourished with arterial blood, providing it with oxygen, salt, etc.

It is quite possible that the frequent evidences of irritation of the auditory apparatus in the way of tinnitus are concerned with abnormal composition or pressure of the fluid in the lateral diverticulum or pathologic changes of the plexus itself.

The author repeats some of the findings noted in his previous paper on disease of the choroid plexus as a whole, and then proceeds to discuss the significance of localized and primarily meningitic lesions of the plexus of the fourth ventricle. In cases of deafmutism, with or without idiocy, pathologic changes appear with greater frequency in the plexus in the lateral diverticulum, leading to atrophic conditions of the neighboring portion of the oblongata and the cerebellum. A third form of disturbance is the result of hypoplasia arising in the earliest embryonal development. A fourth variety represents diffuse and irregular atrophic changes occurring mostly in the plexus in conditions of general arterioselerosis, senility, and renal sclerosis.

Monakow believes that it is not at all improbable that these disturbances may lead to mechanical irritation, resulting in abnormal sensations and leading ultimately to illusions, hallucinations, and schizophrenic phenomena. An analagous condition has been reported on numerous occasions in the sensation of formication, tickling, and the like, occurring in the opposite extremity when the central convolutions are touched in unanesthetized patients. Doubtless, also, noises may result from irritation of the primary acoustic centers, and abnormal sensations of taste from irritation of the glossopharyngeal area. It seems not amiss to venture the statement in the form of an hypothesis that biochemical reactions through the medium of abnormally constituted fluids may evoke responses, similar to those produced by the grosser mechanical irritations of the cortex.

WOLTMAN, Rochester, Minn.

SUR LE PHENOMENE DE STRUEMPELL (STRUEMPELL'S PHENOMENON). NOICA, Rev. neurol. **35**:567 (July) 1919.

Strümpell's phenomenon is briefly described. When the leg of a patient with spasticity is flexed on the thigh there is a flexion of the foot on the ankle, and frequently there is an extension of the great toe not unlike the Babinski phenomenon. The entire sign is more pronounced if the examiner exerts pressure on the knee of the subject without, however, preventing the movement at the knee. Strümpell himself never explained the mechanism of the movement. Babinski and Jarkowski in 1914 regarded the phenomenon as a conjugate movement due to mechanical traction of the aponeurosis in spastic conditions. Marie and Foix, on the contrary, 1916, regarded the sign as due to "active and associated movements" of the anterior leg muscles such as the

vastus externus, the inner part of the soleus, etc.; in brief, the movements of synergic muscles; and as such they regard all so-called conjugate movements, styling them, "syncinetic movements of coordination." These they think are the result of the automatic functioning of the spinal cord freed from the inhibitions of the higher centers, i. e., they are the function of medullary automatism.

This difference of opinion between Babinski and Jarkowski on the one hand, and Marie and Foix on the other, is what stimulated the author to a further study of the Strümpell phenomenon. He made close observation by exerting movements of flexion and extension of the leg and the thigh in the cadaver (after "working out" by frequent movements the rigidity of the joints involved) in a normal boy of 15 and in a normal adult.

In his experiments on the cadaver he confirms Babinski and Jarkowski's opinion that the flexion or extension of one segment (the leg) is accompanied by flexion and extension of the other segment (the foot) and that this is purely mechanical. But he establishes that this phenomenon is not due to any part played by the aponeurosis or tendons of the leg, but aside from the purely mechanical condition, it is accompanied by, or due to, active muscular contraction according to the view of Marie and Foix.

In his experiments with a boy of 15 and with an adult, he comes to the conclusion that we find in normal subjects, especially in young persons, a phenomenon which closely resembles, if it is not identical to, the so-called phenomenon of Strümpell. If, then, a phenomenon analogous to that of Strümpell may be seen in normal persons, and especially in the young, is it equivalent to conjugate movements such as Babinski and Jarkowski conceive them, or is it equivalent to automatic movements of the cord such as Marie and Foix conceive?

The author then analyzes the phenomenon of Strümpell in abnormal or sick persons. He cites the case of a syphilitic patient who had vertigo, double optic neuritis, spasticity in the lower extremities, exaggerated tendon reflexes, sensory disturbances in the feet, lost cutaneous reflexes and a typical Strümpell sign. Yet this patient did not exhibit the great toe sign of Babinski, or the defensive movement of Marie and Foix. That is, there was no automatic cord movement, and yet the Strümpell phenomenon was present. From this it may be concluded that, while usually in spastic conditions, the Babinski, Marie and Foix, and Strümpell signs are all present, yet the latter is not constantly present. The absence of the coexistence of the phenomenon of Strümpell and movements of defense in paraplegics would lead us to think the two phenomena may be different.

The author then emphasizes the condition under which the Strümpell sign is observed; he says the Strümpell sign only obtains in hemiplegics or paraplegics when there exists sufficient muscular power to flex the knee and the foot.

On the basis of observations made on hemiplegics, paraplegics and patients suffering a lesion of the sciatic nerve, the writer regards the phenomenon of Strümpell as distinct from the phenomenon of defense of the physiologists, or the medullary automatism of Marie and Foix.

He adds that he has failed to obtain the Strümpell sign in certain hemiplegics, not because of any pathology in the pyramidal tract, but because of atrophy of the leg muscles in which the atrophy was marked by abundant adipose tissue.

In his final conclusion, the author regards the Strümpell sign as a normal associated movement, modified by the loss of motility of the extension of the leg, and by a partial preservation of the movements of the anterior muscles

of the leg. He also believes that synergic voluntary movements are more easily executed than isolated voluntary movements and that isolated voluntary movements occur later in the development of the individual than synergic voluntary movements. Voluntary isolated movements are brought about by act of will, and then by training; but the will is always necessary, not only to execute the movement, but at the same time to inhibit an associated movement. Therefore, a lesion of the pyramidal tract, which partially inhibits voluntary motility, diminishes at the same time, isolated movements. We revert as it were, to a state of infancy; to a period when we have not yet undertaken to dissociate the complexity of spontaneous movements. We are in a state of being able to execute nothing but synergic movements. Such are the conditions under which the phenomenon of Strümpell exhibits itself.

JONES, Detroit.

SUGGESTIONS AND SUGGESTIBILITY. E. PRIDEAUX, Brain **42**:291 (Jan.) 1920.

Prideaux discusses suggestion and suggestibility. Suggestion is defined as the conveyance and acceptance of an idea without logical grounds on the part of the person who accepts the suggestion. It is largely an affective process, and conviction in the mind of a patient is produced when the suggestion advanced is in harmony with instinctive tendencies, sentiments, interests and complexes and a strong affective force is aroused. This holds for normal suggestion, but is even more pronounced when it is abnormal, as in hysteria. Here the affective currents are stronger and the contrary ones weaker.

Four forms of suggestibility are recognized: First, individual, which varies in different persons, without regard either to the nature of the suggestion or of the suggestor and is explained by the "varying degree in which the egoistic instinctive tendencies are developed, and the manner in which the sentiments have become organized to form ideas and act as contrary forces." Second, conditional, which varies in the same person at different times and under different conditions. This depends "on the relation of the suggested idea to that state." Third, specific, which has reference to a particular system of ideas only. This in itself is proof of the affective nature of the process. It is a well-known fact that a person is particularly receptive to ideas which are pleasing to him and which satisfy his egoistic instinctive tendencies. Fourth, personal, which is effected by the reaction of the suggestee to the suggestor and is more or less dependent on authority, prestige, sympathy, respect, confidence and the like.

The mode of response to suggestion may be classified into positive, negative and neutral. In the positive type the suggestion is accepted not by inhibition of other ideas, but because it becomes endowed with a strong emotional force that overcomes all contrary forces. When the suggestion is opposed, a negative response is obtained. This occurs when the suggested idea is not only "incompatible with preformed sentiments and interests, but arouses contrary emotions and sentiments." The author sees in the negativism of praecox an exaggerated example of such a response. The response is neutral when the suggestion is refused. Here we may be dealing with a person who holds strong principles and ideals or one like an imbecile who has no preformed interests or complexes to which the idea may attach itself and, therefore, the suggestion cannot make sufficient impression, or the suggestion may call forth incompatible forces which neutralize each other.

The author makes some practical psychotherapeutic suggestions. The physician may, and of course as a matter of fact does, use his prestige and authority to stimulate the latent suggestibility of the patient. If the patient is only suggestible in a particular sense, then the suggestions must be arranged and presented so that incompatibilities will be avoided. Favorable effective states may be created. Thus surprise will "clear the mind" and make a response more likely. If fear exists it may be neutralized by inducing anger, for these two emotions "tend to exclude one another from simultaneous activity."

STRECKER, Philadelphia.

SUICIDE AMONG SOLDIERS RECEIVED AT THE HOSPITAL FOR PSYCHIATRIC CASES AT MOMBELLO. GIUSEPPE SACCHINI, Arch. di antrop. crim. **29**: Pt. 2.

A statistical study of the soldiers received at the psychiatric hospital reveals the fact that suicidal attempts are more frequent among mentally diseased soldiers than among civilians. War, by its violent reactions, renders less repugnant and even familiar the idea of death. For one type of person suicide becomes the preferred remedy for all his anguish.

Detailed histories of fifty cases are given. In only one case was the result fatal. Fourteen of the men had previously attempted suicide in civil life. The hereditary taint among the fifty patients was marked, alcoholism being present in 32 per cent., suicide in 30 per cent., psychopathy in 16 per cent., neuropathy in 16 per cent., etc. In only four patients was there no taint. Ten were syphilitic and three tuberculous, while in three others the patient feared one or other of the above diseases. Six patients were convalescing from a trauma or acute disease while in the past history of twenty others there was a history of serious illness followed by neurasthenic states, intermittent headache, irritability, weakening of the critical power, etc. The exciting cause of the suicidal attempt was "tired of living" in eight patients; hypochondriac preoccupations in nine; disappointment in love in four; and in six there was no assignable cause. Among the definitely pathologic causes were: attacks of unconsciousness in seven, brief and partial blurring of consciousness in five and grave mental disturbance in three. Among the strictly military reasons were fear of censure by superiors and fear to return to the front. During their convalescence eighteen were in a melancholic state and fifteen others showed definite mental pathology. The predominating forms of mental disturbance were neurasthenia, epilepsy, dementia praecox and feeblemindedness. As to the means of committing suicide, twenty-two chose poison, four fire-arms, five cutting instruments, nine threw themselves to the ground from a height, five threw themselves into the water, two under heavy vehicles and seven attempted hanging. Two made two or more attempts by one or more methods. Seven had previously been refused for military service, eight had criminal records and almost all showed degenerative stigmas. The recruiting of such defectives naturally markedly increases the percentage of all crimes among soldiers.

The necessity of foreseeing the consequences of congenital and acquired mental abnormality has been widely recognized in this war with a resultant increase in the military hospitals for the observation and cure of alienated soldiers.

The article is a careful analysis of the conditions leading up to attempts at suicide and an argument for the abnormal basis in the vast majority of cases. It is also an argument for the more careful selection of recruits, a lesson learned early in the late war by the United States military authorities.

VAN EPPS, Iowa City.

LE TARTRATE BORICO-POTASSIQUE ET LA MEDICATION BOREE DANS LE TRAITEMENT DE L'EPILEPSIE (BORIC POTASSIUM TARTRATE AND BORON MEDICATION IN EPILEPSY). PIERRE MARIE, CROUZON and BOUTTIER, Bull. de l'Acad. de méd. **83**:504 (June 1) 1920.

Thirty years ago Gowers extolled the use of borax (sodium borate) in epilepsy. Chemical impurities (?) of this drug have rendered results with it poor or uncertain. Therefore the authors have experimented with other boron salts using chiefly boric potassium tartrate, otherwise known as boric emetic and also as soluble cream of tartar; with the following atomic formula: Co_2H-CHOH-CHO (B_o O)-CO_2K, and have watched the effect of this drug in gram doses three times a day on patients at Salpêtrière, who were "simple epileptics"—i. e., nonalienated They have used sodium tetraborate in other series.

They chose ten female epileptics who showed attacks maximal in frequency and intensity. For the months that the drug was administered and for the month preceding these patients were carefuly observed, day and night, so that all attacks might be recorded. (They had all been without bromids for either years or months.) The apparently beneficial effects of the drug is shown by the fact that these ten patients during the month preceding the administration of the drug averaged thirty and one-half attacks per month each, while during the period of treatment, three months, they averaged eighteen attacks each during the first month, eleven and one-half during the second and nine during the third. The writers not only report benefit in that the attacks were reduced in number but a greater portion of the attacks were vertiginous in nature rather than convulsive.

The drug is well borne and the occasional gastric disturbance encountered (diarrhea and vomiting) respond to its temporary withdrawal. Especially conspicuous is the total absence of depressive action on the psyche. Acne has been induced in some cases but not in a degree comparable to that caused by the bromids. Equally satisfactory effects have been obtained in children between 7 and 15 years of age. The authors have obtained benefit even in traumatic epilepsy and in epilepsy caused by cerebral tumor.

The therapeutic effects of bromids have especially for their basis the diminution of the reactional power of the nerve centers. For boric potassium tartrate it seems to be otherwise. One has the impression that it is on the lesion which is causing the epilepsy that the effect is gained. One is tempted to ask whether it is not by a sort of catalytic action that a small dose of boron determines such notable effects.

The conclusions are: 1. Boron medication plays an anti-epileptic rôle quite equal to that of bromin medication. 2. Boric potassium tartrate is superior to the other boron salts for the purpose.

DAVIS, New York.

CRIMES DUE TO THE WAR NEUROSES. Prof. Leone Lattes, Arch. di antrop. crim. **29**: Pt. 1-2.

The war neuroses as efficient causes of criminal acts have not been given sufficient recognition in comparison with other psychopathic states. After excluding all the cases in which "shell-shock" has been used as an excuse there remains a definite number which may be termed the "crimes of the psychoneurotics." These merit a more general consideration than commonly accorded because delicate questions of justice, humanity and social prophylaxis are involved. The difficulty arises not during the acute symptoms, but later when the convalescence is incomplete.

A detailed history is given of fourteen cases. In one the crime was homicidal attempt, in four desertion and in nine insubordination. Of the above cases two gave a history of morbid heredity and two of previous morbid symptoms. The relations between the crime and the neurosis is one of dependence and not of concomitance. The personality has been changed so that crime becomes possible. In almost all of the patients the previous conduct had been good and occasionally even distinguished. Only one had a previous military sentence and none a civil record. The typical offense of the psychoneurotic is insubordination based on a morbid irascibility and lack of inhibition. As the result of some innocent remark or reproof the patient reacts excessively and loses self control. The clinical evaluation of these facts is simple; the medicolegal is more difficult. The difficulty arises in the fact that the crime is dependent on a morbid alteration, the result of the injury to the mind and not on a constitutional psychic anomaly. In the interpretation of the code the morbid state of the accused at the time of the act nullifies the accusation. The military code is essentially preventive in scope. But rising above the restrictive view of punitive practice is that of social preservation. No doubt the symptoms of the psychoneurotic may be confused with criminal tendencies for the responsibility is intact, but the opposite mistake is more frequent and more embarrassing. The remedy is mainly preventive, namely, the recognition of the war neuroses and the establishment of cure before return to duty. This, however, demands an individualization in treatment and a stability of judgment rare in the vortices of war.

Van Epps, Iowa City.

LE POLYMORPHISME DE L'ENCEPHALITE EPIDEMIQUE, CLASSIFICATION DES FORMES CLINIQUES (THE POLYMORPHISM OF EPIDEMIC ENCEPHALITIS WITH A CLASSIFICATION OF CLINICAL FORMS). Henri Roger, Progrès méd. **35**:247 (June 5) 1920.

The author first points out the polymorphism of epidemic encephalitis and this is sufficiently emphasized by the classification which he then proceeds to formulate.

Apart from distinctions based on the soil in which the disease evolves (infantile form, senile form, gravid form) or apart from distinctions resting on anatomy (peduncular, bulbar, spinal or even polyneuritic forms) or apart from distinctions based on the course (acute, subacute, chronic, ambulatory or relapsing forms), he proposes, on a clinical basis and following the methods of Grasset, to distinguish forms due to exaggeration, deficiency or vitiation of nervous system functions: in other words, types in terms of hyperfunction,

hypofunction and parafunction. On this basis he makes the following classification:

1. Motor Forms:
   (*a*) Hyper: myoclonic, choreo-ataxic, convulsive, tetanic.
   (*b*) Hypo: paraplegic type (of Sicard), polyneuritic (variations and combinations under (*a*) and (*b*) introduce the syndrome Millard-Gubler form, the oculomyochonic form, the ophthalmoplegic form).
   (*c*) Para: Parkinsonian or myotonic.
2. Sensory forms "of which we know only the hyper type, the algic."
3. Psychic forms.
   (*a*) Hyper: delirious, mental.
   (*b*) Hypo: with psychic depression.
4. Forms resting on the functioning of the sleep center.
   (*a*) Hyper: somnolent or lethargic.
   (*b*) Hypo: insomnious.

DAVIS, New York

PSYCHOSIS ASSOCIATED WITH TETANY. ALBERT M. BARRETT, Am. J. Insan. **76**:373 (April) 1920.

Barrett had the unusual opportunity of studying two cases of tetany both of which came to necropsy. Two men, one aged 58 and one 46, presented classic neurologic symptoms of tetany. Mentally there were varying degrees of clouded consciousness at times amounting to stupor, depression and apprehension, auditory and visual hallucinosis. Of great interest is the fact that these symptoms appeared at irregular intervals and were succeeded by comparatively clear periods. In one case the relationship between an increase in the mental disturbance and constipation with excessive indican is significant.

Postmortem studies showed the effect of intense toxicity on the brain and cord. In both cases the pituitary body and thyroid were cystic. In one instance one parathyroid was independent, and there was parathyroid tissue in the substance of the thyroid. In the other three parathyroid were found. In neither case were there pathologic changes in these glands.

Both of Barrett's cases had numerous cysts of the pituitary glands. Here is additional evidence of the connection between these bodies and the parathyroids. It has already been noted that the hypophysis enlarges after thyroidectomy and parathyroidectomy, and tetany symptoms consequent on parathyroid removal in cats have been made to disappear by pituitary injection.

The author concludes that there is no specific tetany psychosis but that both the neuromuscular and the psychotic syndromes result from a toxic process affecting the central nervous system.

STRECKER, Philadelphia.

Society Transactions

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Meeting, May 20, 1920

E. W. Taylor, M.D., *President*

REFLEX PHENOMENA IN THE CONTRACTURE STAGE OF PERIPHERAL FACIAL PARALYSIS. Presented by Dr. A. Myerson.

Following the acute phase of peripheral facial paralysis, of whatever origin, there is in the cases that partly recover a phase of contracture. In this period, which comes on gradually after six months or more, the patient superficially presents a picture quite the reverse of the acute phase in that the nasolabial fold on the affected side is more prominent and the aperture of the eye is narrower. There is usually some weakness of the face, with so-called associated movements, so that when the affected eye is closed the corner of the mouth moves with it.

The new phenomena described (illustrated on a patient) are: (1) when an electrode (either G. or F.) is placed on the affected brow and the current turned on, the corner of the mouth twitches before the brow contracts, although the direct excitability of the nerve is lessened; (2) tapping the forehead or the bridge of the nose causes a contracture (reflex) of the affected corner of the mouth.

These phenomena are absent on the sound side and in all other conditions. They seem to be specific to the contracture phase of the peripheral facial paralysis and relate to changes in the nucleus whereby excitability is increased and made diffuse.

THE DIFFERENTIAL DIAGNOSIS OF HYPERTHYROIDISM. Presented by Dr. J. H. Means.

Overactivity of the thyroid gland is of interest to the neurologist, not only because of the numerous clinical nervous manifestations to which it gives rise, but also from the point of view of functional pathology.

The work of Cannon and his collaborators has clearly shown an intimate relationship between the thyroid gland and the sympathetic nervous system, also between the suprarenal and the thyroid. Secretory activity of the latter can be shown to occur if the sympathetic nerve is stimulated or if epinephrin is injected, and augmentation of the pressor action of epinephrin takes place on a sympathetic stimulation. Furthermore, a condition in many respects like exophthalmic goiter has been produced in cats by the anastomosis of the phrenic with the cervical sympathetic nerve.

The work of L. B. Wilson is also interesting in this connection. This investigator found in the cervical sympathetic ganglions, which had been removed from patients with exophthalmic goiter, definite lesions of the ganglion cells which he regarded as causally related to the morbid process in the thyroid.

The fully developed case of exophthalmic goiter presents no difficulties in the matter of diagnosis. The neurologist, however, is frequently confronted with patients presenting atypical or suggestive symptoms of hyperthyroidism which he may experience difficulty in interpreting. The differential diagnosis of the milder grades of hyperthyroidism may be no easy matter. As a rule, the differential diagnosis will be from various psychoneuroses, cardiac neuroses, effort syndrome, etc., and from early tuberculosis.

Of late years certain rather technical laboratory tests have been helpful in differentiating true hyperthyroidism from other types of disorder. Probably the most useful of these is the determination of the so-called basal metabolism. One of the chief functions of the thyroid gland is to preside over the rate of heat production of the animal body. An increase in heat production occurs in hyperthyroidism and a decrease in hypothyroidism. The determination of the combustion level, therefore, serves as a functional test of the thyroid. As such it is useful not only in estimating the relative degree of hyperthyroidism in a case of exophthalmic goiter, but it also serves as a means of differential diagnosis in borderline cases, the metabolism being elevated in true hyperthyroidism and not in the other disturbances with which hyperthyroidism may be confused.

The level of the heat production or basal metabolism can now be easily determined in the clinic by means of a respiration apparatus by the method of so-called indirect calorimetry.

The reaction to epinephrin has also been used to detect the presence of hyperthyroidism. It is undoubtedly positive in all cases of true hyperthyroidism, but, as Peabody has recently shown, it is also positive in such conditions as effort syndrome. It is therefore less reliable than the determination of the metabolism level as a diagnostic procedure.

The blood sugar curve has also been suggested. Aub and Davis, however, have shown that while an alimentary hyperglycemia is readily produced in hyperthyroid patients, the height of the curve bears little relationship to the intensity of the thyrotoxicosis.

In general we must regard these special tests as helpful, but we must, as in everything else, place the information gained by our five senses first. Nothing can supplant the importance of the clinical impression, but the special tests, as Dr. F. C. Shattuck has said, if we make them our slaves and not our masters, will be of distinct aid.

(The clinical records and charts of three patients which illustrated the value of metabolism studies both in differential diagnosis and in following the progress of exophthalmic goiter were then presented.)

CARDIAC NEUROSES. Presented by Dr. Paul D. White.

Cardiac neuroses are functional disorders of the nervous system attended primarily by cardiac symptoms. This subject is as old as the study of medicine itself; Hippocrates, Galen, and nearly all the other ancient masters wrote of it. It becomes obscured now and again and constantly needs reemphasis.

The recent war revealed the fact that the irritable heart of soldiers is of frequent occurrence. It is essentially a neurosis of the fatigue or anxiety type and should not be considered as heart disease. The frequency of its occurrence in the army was such that Dr. White's attention had since the war been called to its frequency in civilian life, particularly among young women. It seemed

that nearly one-third of all the so-called "cardiac" patients under 40, whom he had been seeing, belong in this large group of cardiac neurotics. Obviously, therefore, the subject is very important.

The differential diagnosis of cardiac neurosis includes consideration of true heart disease, hyperthyroidism, low grade infections like tuberculosis and convalescence.

A working classification built on that of the psychoneuroses presented by Dr. Stanley Cobb is as follows:

1. Fatigue neurosis—so-called "neurasthenia."
2. Introspection neurosis—so-called "hypochondria."
3. Anxiety neurosis.
4. Substitution neurosis—so-called "hysteria."
5. Obsession neurosis—so-called "psychasthenia."

Ninety-five per cent. of all cases.

DISCUSSION

DR. MORTON PRINCE asked whether Dr. White had found any enlargement of the heart accompanying his cases of cardiac neurosis.

DR. PAUL D. WHITE replied that patients who had cardiac neurosis pure and simple had no enlargement nor murmur. There was no sign of actual heart disease in any case of true uncomplicated cardiac neurosis.

DR. MORTON PRINCE said that he had examined the hearts of many thousands of men in the prime of life and found not only increased heart action (sometimes irregular), but also an enlargement of the heart. In a good many cases mitral systolic murmurs were present. Not being certain of the organic origin of these symptoms, Dr. Prince made a long series of investigations on several hundred men with the result, as was demonstrated, that he found the cardiac symptoms were due to the emotional excitement attending the examination. It became plainly evident that the enlargement of the heart, when present, was only the normal physiologic dilatation that occurs under excessive stimulation to enable the heart to do the work that it is called on to perform. In some cases the dilatation was so extreme as to permit some mitral valve leakage, thus causing the murmurs.

As a control experiment, a series of examinations was conducted under conditions in which the emotional excitement was eliminated, with the result that the previously observed cardiac symptoms disappeared. In another series of observations the anxiety was artificially stimulated as much as possible: in this series the symptoms were even more frequent. These observations were published in the *Medical Record* and the *Transactions of the Association of American Physicians,* and were among the first reports to show that the heart expands and contracts according to the work to be done and that this occurs under nervous stimulation.

The disturbances observed were of a temporary nature, but they are comparable to those observed in an anxiety neurosis of a chronic character. They might be called "acute anxiety neurosis." They are in principle what used to be called "irritable heart" when the emotional factor was overlooked. They should be considered not as the manifestations of an abnormal heart, but as the normal physiologic cardiac expression of abnormal anxiety. The anxiety is pathologic, not the cardiac manifestations.

Dr. A. Myerson asked Dr. Means whether in nontoxic goiters the metabolism varied. Dr. White brought up the point that cardiac neuroses were found in young people. Why are they not found in older people? Do such patients die, and if they do not spontaneously recover, how do they get well? Reassurance in his experience has little value. Does the condition disappear with early adult life and what is the mechanism of recovery?

Dr. Walter B. Swift said that a word ought to be said about the speech in cases of goiter. In 1918 a goiter zone was discovered in Cleveland and the speech studied in thirty cases. Each one of these patients was put through a series of some forty speech tests, making in all over 1,200 speech tests with tabulation of data on each. He was not ready to give full details of this paper, but investigation showed that goiter speech as a whole is diminished, slightly slow, of remarkably low pitch and harsh. In lay terms it might be called mannish when it occurs in women, whose natural speech is an octave higher than speech in men. In contradistinction, and as an item of differential diagnosis, the speech in neurasthenia is inclined to be fuller than usual in amount, increased in speed, high in pitch and rather quick and reflexlike in nature. The cause could not be found at first.

Dr. Morton Prince said that he had a strong suspicion that in many of Dr. White's cases anxiety was present. This is easily overlooked, but can be brought out readily by searching examination.

Dr. James B. Ayer said that he understood that when Dr. Goetsch returned from Saranac he said that about half the patients treated there for incipient tuberculosis were, in his opinion, cases of hyperthyroidism. It would seem to him reasonable that the asthenia of the tuberculous patient, coupled with temperature, might make a particularly susceptible sympathetic nervous system which would respond to the suprarenal test, giving the picture of hyperthyroidism. Dr. Ayer asked Dr. Means whether he still considered that there was a metabolic change which is characteristic of hyperpituitarism and hypopituitarism as well as hyperthyroidism and hypothyroidism.

Dr. W. E. Paul said in regard to thyroid changes that the hyperthyroid cases may become hypothyroid as time goes on. He had seen one or two and would be glad to hear of any others.

Dr. Henry Viets said that some one had suggested that in cases of encephalitis lethargica sleepiness may be due to some change in the pituitary gland. He wondered whether Dr. Means knew of any relation of metabolism to the marked lethargy of lethargic encephalitis.

Dr. Donald Gregg asked how it was possible to tell whether there was not some suprarenal disturbance in some cases in which a positive reaction with epinephrin was obtained. The thyroid disturbance is barred out, but how about the suprarenal cases? Do they also react very strongly?

Dr. J. H. Means, answering Dr. Myerson's question about nontoxic goiter, said metabolism is always normal in this condition. He thought there were two fairly definite diseases—exophthalmic goiter and adenoma. Exophthalmic is a uniform hyperplasia of the thyroid gland with hyperthyroidism. Any adenoma may apparently become toxic; then we may have a hyperthyroidism. Under those circumstances there is an elevation in the metabolism, but it is always accompanied by clinical manifestations. Goiter patients who are perfectly well except for the lump in the neck have no elevation.

Answering Dr. Ruggles' question about myxedema, Dr. Means said in general the clinical picture runs about parallel with the fall in metabolism. The fall is usually about from 25 per cent. to 33 per cent. The metabolism level is an indication of the amount of thyroid to give. He thought the dose required in individual cases varied a good deal. For example, he cited the case of a nurse who had an acute thyroiditis, an infectious process. She had had a nontoxic goiter for a number of years. It suddenly increased in size and became very tender, and she had a high temperature. The gland was removed surgically and on examination showed a definite inflammatory process—not exophthalmic goiter. About three weeks later Dr. Means found the metabolism was 15 per cent. The patient looked normal. He saw her again about four weeks later. In the meantime, she had developed typical myxedema. The drop in metabolism had preceded the development of clinical symptoms. After the administration of thyroid she became normal.

Answering Dr. Ayer's question, Dr. Means said that he had no personal experience with the Goetsch test. He believed that some of the cases that reacted to his test were not cases of hyperthyroidism, but were some kind of neuroses. He believed the thyroid glands were not abnormal, but that the vegetative nervous systems were unduly active, which is fairly distinct from true hyperthyroidism. Regarding pituitary conditions, there is a change in metabolism in pituitary disease. It is not so constant or so striking as in hyperthyroidism, but in active cases of overfunctioning of the pituitary gland there is sometimes an increase of about 40 per cent. and in some cases of hypopituitarism a slight reduction. There is no definite alteration in old acromegalics. When the process has died down the metabolism is generally normal.

Dr. Means answered Dr. Paul's question in the affirmative because recently he had studied a case of mild exophthalmic goiter. The patient's rate was + 50 per cent. only six weeks ago. She had all the symptoms of exophthalmic goiter. After three roentgen-ray treatments she came back to the hospital and he didn't recognize her. She presented a picture not characteristic of myxedema, but suggestive of it, and her metabolism was 12 per cent. below normal. She was given small doses of thyroid and became more nearly normal in appearance. This is the only patient he had ever seen who became definitely myxedemic after roentgen-ray treatments. Some of the postoperative cases that he had seen suggestive of myxedema had a slightly depressed metabolism.

Answering Dr. Viets' question, Dr. Means said he did not know of any observations that had been made on lethargic encephalitis.

Dr. Means could not answer Dr. Gregg's question. We know very little about primary disease of the suprarenals. He had one case of Addison's disease which showed no change. Cases of recognized suprarenal deficiency are very rare.

Dr. Paul D. White agreed with Dr. Prince except in regard to the effect of excitement on the heart size. The term "soldier's heart" is satisfactory. Anxiety undoubtedly plays a more prominent part than we realize. He recently saw a patient in whom the anxiety neurosis was undoubtedly a factor—a young woman with a disfiguring lupus vulgaris. She had typical cardiac neurosis symptoms. The effect of exercise and excitement on the heart is, of course, to increase the rate and force of the heart beat and, if the case is extreme, one also

finds the presence of a systolic murmur. Immediately after exercise the heart is actually smaller than before, as proved by the roentgen-ray examination. The force of the heart is such that, on physical examination, the apex impulse may give an impression of dilatation. The murmur is commonly found at times of excitement, probably not to be explained however by dilatation of the mitral ring. His experience was too limited to tell what happens to these patients with cardiac neuroses as they grow old. They probably recover. An important point may be the relationship of cardiac neurosis to a latent production of arteriosclerosis. It is possible that one of the factors of arteriosclerosis is the constant strain which accompanies these symptoms in youth. The Goetsch test is very much like the reaction of excitement.

Book Review

ETUDE SUR L'EPILEPSIE TRAUMATIQUE. Dr. Pierre Béhague, Médecin aide-major de 1re classe au Centre neurologique de la Salpêtrière. Travail du Service de la Clinique des maladies du système nerveux à la Salpêtrière (Prof. Pierre Marie), Louis Arnette, Paris, 1919, 320 pages.

This unusually good thesis is from the "militarized" service of Pierre Marie at the Salpêtrière, where of 3,623 patients with head injuries, 439 are known to have developed epilepsy. These cases form the basis of the present study. Owing to admirable cooperation between the neurosurgical service and the neurological service, and to a rule requiring patients to report at the hospital from time to time after discharge, the observation of a large proportion of the patients was unusually complete. In other words, the enormous material was wisely utilized and the result is a most valuable composite picture of traumatic epilepsy. The work is divided into three parts: I. Immediate and transitory epilepsy. II. Established epilepsy (épilepsie durable). III. Late symptomatic epilepsy.

I. These convulsions, which may be local or generalized, are caused by concussion injury, by foreign bodies or by hemorrhage, and soon disappear, or status epilepticus develops with fatal outcome unless the hemorrhage is surgically arrested. A little later convulsions may be caused by septic process.

II. This group of convulsions is due to concussion, obvious injury of the brain or to extracranial trauma which interferes with the cerebral circulation (injury or ligation of large vessels of neck).

It is worthy of note that the author found no case of so-called reflex epilepsy due to injury of a peripheral nerve.

One of the most instructive showings of the analysis of these obviously traumatic cases was that only one-third of the patients had focal paroxysms—motor, sensory or special sense—while two-thirds had generalized convulsions of the idiopathic type. In four-fifths of the cases the injury was either of the parietal (55 per cent.) or frontal (26 per cent.) region, but a few were caused by basilar fracture with no injury of the vault, and a few by concussion with no fracture and very little external injury. The analysis includes the relation of location of the trauma to character of the convulsions, the most various of brain injuries, the period of latency and many other clinical features. Like other investigators, the author found that syphilis and alcoholism predispose to traumatic epilepsy, that preexisting epilepsy is aggravated by brain trauma and that traumatic epilepsy is aggravated by subsequent injury. Most of the wounds causing epilepsy were septic.

The author's opinion on surgical intervention in head wounds is interesting. Surgery of the cranium alone is unimportant. But, early or late, operation on membranes or brain necessarily increases the already existing damage and hence favors the birth of epilepsy. Also of interest is his opinion of cranioplasty. He is sure that it may cause epilepsy; he found 22 cases in 99 cranioplasties. In those already epileptic the operation increases the convulsions or has no influence. He is also opposed to operation for cerebral hernia.

The latent period is shorter in the jacksonian type. Danger of epilepsy is greatest in parietal wounds; frontal, occipital and temporal wounds follow in

the order given. The danger increases with the size and inversely with the velocity of the projectile; with the depth of the wound and especially with the extent and number of surgical procedures and the presence of suppuration.

The clinical descriptions are excellent, from prodromes and auras through different types of seizures to various epileptic equivalents. Diagnosis is adequately covered. There are twelve pages on pathogenesis with the following rather sterile conclusions:

1. Epilepsy is always caused by cortical irritation. This can be caused by a cicatrix, foreign body, tumor, etc., but especially by neuroglia cicatricial proliferation of hemorrhagic or traumatic origin.

2. The irritation is caused principally by expansion of the brain against the irritating substance.

3. This expansion is the result of ventricular dilatation due to increase of arterial tension which is facilitated by the peculiar structure of the choroid plexus.

4. The hypertension of the cerebrospinal fluid is secondary and can cause only generalization of the convulsion.

Regarding surgical treatment, Béhague endorses the conclusions of the interallied surgical conference, January, 1918. In brief these are: Jacksonian epilepsy may be benefited by removal of a foreign body, splinters, etc. The operation should be limited to removal of cortical irritants and no search made for deep seated bodies. Operations for other types of epilepsy are condemned. In medical treatment nothing new is offered.

III. By late symptomatic epilepsy the author means convulsions coming on after the head wound has healed, as a result of some complication, such as abscess, hemorrhage or nonsuppurative encephalitis. Such convulsions appear suddenly, are frequent and violent and demand immediate attention. An excellent microscopic study of two cases of nonsuppurative encephalitis closes this section.

The monograph concludes with notes of 444 cases, 13 formal conclusions and 22 pages of bibliography. It is a fine piece of work, valuable to neurologist and surgeon alike.

The American Medical Association will pay 50c each for the April and May, 1919, issues of the ARCHIVES OF NEUROLOGY AND PSYCHIATRY. Address to American Medical Association, 535 North Dearborn St., Chicago, Ill.